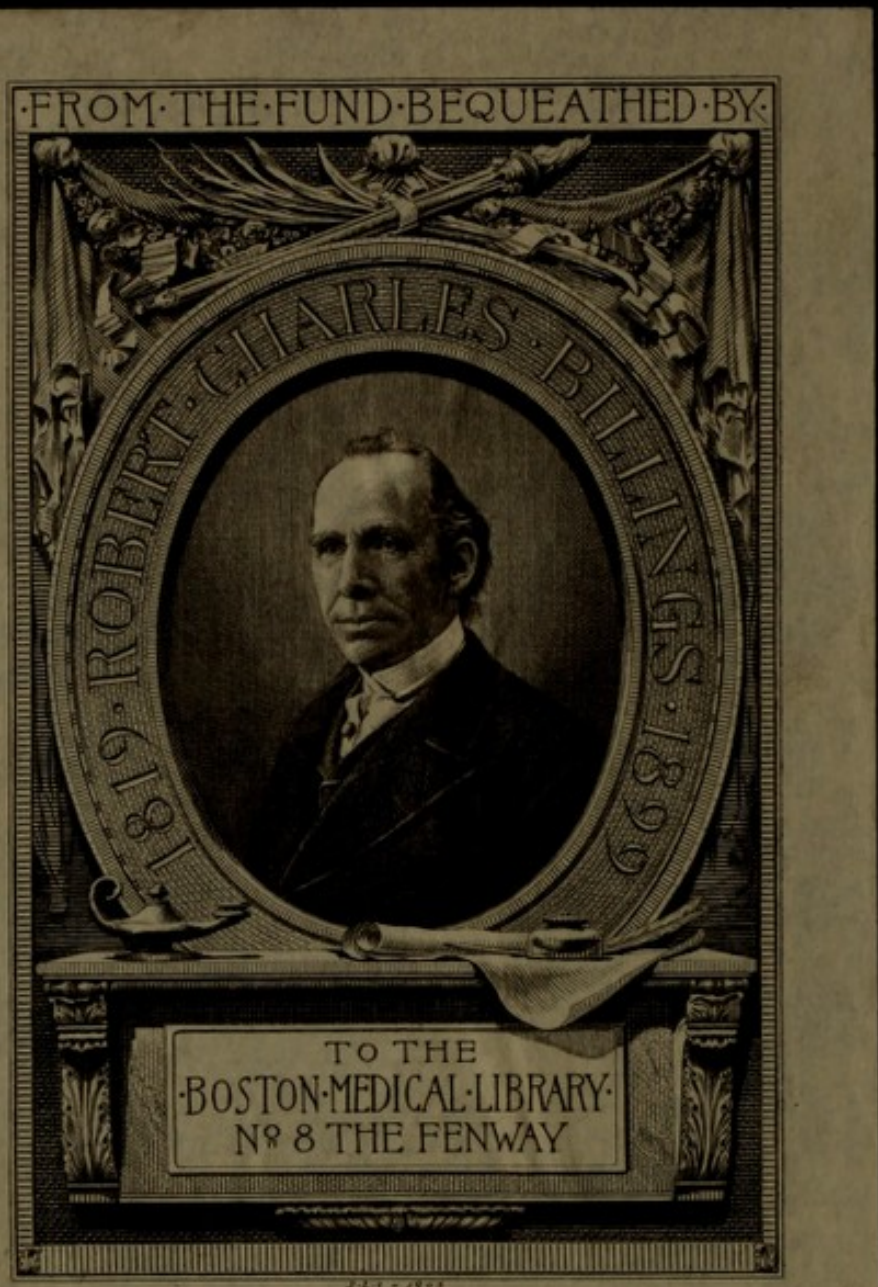
The prevention of valvular disease of the heart : a proposal to check rheumatic endocarditis in its early stage and thus prevent the development of permanent organic disease of the valves / by Richard Caton.

THE PREVENTION OF VALVULAR DISEASE OF THE HEART

R. CATON M.D. F.R.C.P.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THE PREVENTION

OF

VALVULAR DISEASE OF THE HEART

A PROPOSAL TO CHECK RHEUMATIC ENDOCARDITIS IN ITS EARLY STAGE AND THUS PREVENT THE DEVELOPMENT OF PERMANENT ORGANIC DISEASE OF THE VALVES

BY

RICHARD CATON, M.D., F.R.C.P.

HON. PHYSICIAN LIVERPOOL ROYAL INFIRMARY,
EMERITUS PROFESSOR OF PHYSIOLOGY, UNIVERSITY COLLEGE, LIVERPOOL.

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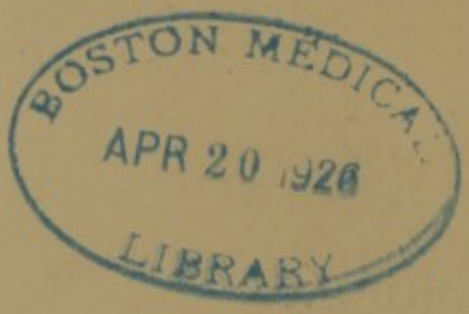

TO

LORD LISTER,

PRESIDENT OF THE ROYAL SOCIETY,

WHOSE WISE APPLICATIONS OF SCIENTIFIC
PRINCIPLES FOR THE LESSENING OF MORTALITY AND
SUFFERING AMONG MANKIND
ARE UNEXAMPLED
IN THE ANNALS OF MEDICINE,
THIS BRIEF TREATISE
IS BY PERMISSION
DEDICATED
BY

THE AUTHOR.

PREFACE.

IT is the desire of the author to state in this volume as clearly and in as few words as he can the results of an enquiry in which he has been engaged for the last nineteen years, as these results appear to him to have some value and interest.

Valvular disease of the heart has always been and still remains one of the opprobria of medicine. It is a serious cause of suffering to mankind, and will probably always continue to be incurable.

The author's object has been to promote the detection of this malady in its early stage; to study the commencing pathological changes in the valves; to investigate the manner in which in a few cases a natural arrest of the disease is brought about; and to seek by artificial means to render this arrest in the early stage usual, rather than exceptional.

Details of the method proposed are given, and the theory on which it is founded is explained.

Brief notes are given of eighty-six cases, successful and unsuccessful, in which the method advocated has been tried.

The Author is indebted to his colleague Dr Hill Abram for important assistance in the pathological part of the enquiry.

August, 1900.

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CHAPTER I.

THE FREQUENCY AND THE GRAVITY OF VALVULAR DISEASE.

No physician who has had a lengthy experience of hospital or private practice can fail to be impressed with the number and the seriousness of the cases of organic heart-disease which come under his notice. He cannot but be saddened by the enfeebling and shortening of valuable lives which occur from this cause, and particularly so when the crippling of the heart happens, as it so commonly does, early in life, and the sufferer, in place of keeping step with his fellows, is compelled to fall out of the ranks, and not only abandons most of the hopes which brighten life, but even experiences the loss of all power to earn his daily bread. If the patient's position in a pecuniary point of view is a good one, the prospect is not so unfavourable, much may yet remain for him to look forward to, he may be able to avoid all the muscular stress which is so destructive to a diseased heart; but if he be a poor man who has only the labour of his hands to depend upon his prospect is a melancholy one, for every exertion made in earning his livelihood is but inflicting a further injury upon an enfeebled heart and bringing nearer the period of

its inevitable breakdown. The evil is so great that the author has often thought that some public provision should be made for these cases which lie in the dilemma between starvation or heart-failure. Too often the labouring man whose heart is giving way under the strain comes to the physician with the piteous question, "Doctor can you not cure me?" and he to whom the appeal is made is obliged in some degree to dissemble; he gives what advice, encouragement, and temporary help are possible, but he cannot be so cruel as to say there is no hope, though he knows with only too great certainty how unfavourable the issue must be.

In this country out of every million people living, nearly seventeen hundred per annum at present die from circulatory disease: from this fact we may glean a partial indication of the years of weakness, disablement, and suffering which precede the fatal issue occasioned by these maladies.

We have no means of repairing a destroyed cardiac valve: when a mitral cusp has become altered and shrunken, has undergone necrosis or calcification and cannot quickly oscillate with the flux and reflux of the blood-current, or when adhesions have formed between adjacent cusps, narrowing the channel through which the stream on which so much depends has to flow, when these morbid changes are finally organized and established, we know of no method that can fully compensate for the mechanical defect. Nor does it appear probable that any will ever be found: we can of course give valuable temporary help and relief, and the natural powers supply for a time what we technically call "compensation," but it seems highly

improbable that the reinstatement of a structurally abnormal heart in its original physiological perfection as a machine can ever be effected, however far our knowledge and our therapeutic powers may advance in the future.

If it be true that the disease is so serious, and that cure in its strict sense is impossible, preventive measures are highly important, if prevention be in any degree practicable.

The causes of endocarditis.

In view of the question of prevention, the study of the causes and of the early stages of endocarditis becomes important. The chief antecedent causes we know with considerable accuracy. Acute and sub-acute rheumatism, and those forms of this disease characterised by chorea, cutaneous nodules, the so-called "growing-pains," and the tonsillitis of childhood, are the most important; in minor degrees other septic conditions, such as enteric fever, scarlatina, pneumonia, erysipelas, influenza, and tubercle. Micro-organisms, among which are staphylococci, pneumococci, or streptococci are almost invariably present in the structures affected.

The number of different varieties of these organisms which have been described by bacteriologists of late, such writers for example as Letulle[1], raise some doubts in one's mind as to how far the microbe is to be regarded as the cause or merely as an accidental concomitant, and also whether much is to be gained in the future from the use of antitoxins, towards which we have been looking with hope.

[1] *Anat. Pathol.* pp. 112, 113.

It is when treating the ailments above named that we must be on the watch for the early symptoms of valvulitis, if there be any hope of preventing the disease or of averting it while in the incipient stage.

The possibility of preventing or arresting valvulitis.

The great primary question we have to consider is, Have we any evidence that prevention or arrest is practicable? Is it worth while to make any attempts in such a direction? Must the practitioner be content always to remain a mere passive spectator of this the most formidable and fatal phenomenon of acute rheumatism? Is it after all so certain that no means of prevention exist?

In reply to these questions let me remark that early endocarditis accompanied by all the signs of mitral or aortic regurgitation does unquestionably, in a few cases, subside naturally, leaving no evil results, without any aid from treatment. I personally have seen this happen, and it would not be difficult to adduce abundant corroborative evidence from authorities of the highest credibility.

Though it is the unfortunate fact that this spontaneous cure seldom occurs, still the truth remains that nature can and occasionally does completely restore a damaged valve, in the early stage; and in this truth I think there is found much hope and promise. Ought we not to study carefully the lesion itself and its various stages of development, so that we may comprehend up to what point in its downward progress the affected valve is curable, the length of time which elapses before complete and hopeless

destruction takes place, and the conditions which appear to favour recovery?

Only the natural powers can effect restoration; but can we not so modify the conditions in which the circulation and also the patient in other respects is placed as to make the process of restoration easier? Can we not remove difficulties and supply aid and stimulation to the natural powers, so that repair and recovery may be more frequent? Can we in no way help nature to produce as complete a cure in the heart as in the joints?

This is the problem which in the earlier part of this little treatise is to be considered.

At the present time most members of the medical profession consider all effort at prevention or arrest of valvular disease as a hopeless task.

Most of our order regard the matter in a somewhat fatalist spirit; if the rheumatic patient is assailed by valvulitis we lament the occurrence, we hope it may possibly subside, though we know it usually does not, and we in general conceive ourselves absolutely powerless to influence it. Personally I confess this was my own belief and practice during the first twelve years of professional life: but nevertheless, nineteen years of subsequent experience, devoted especially to the study of this question, have securely founded the conviction that much may be done to prevent the mischiefs of valvulitis and also to arrest the disease when it has only lately commenced. So clearly indeed has this truth become evident to my eyes, that I should now feel it a distinct dereliction of duty on my part if I disregarded certain precautions and certain methods of treatment in each case I meet with.

Professional reluctance to accept new methods.

In the acceptance of new proposals of treatment our profession observes a cautious scepticism. On the whole this is a wise mental attitude. But we should remember Lord Beaconsfield's dictum, "*Incredulity* also has its dupes." The writer when a young house-surgeon in a great hospital saw the introduction of antiseptic surgical practice; he cannot easily forget the almost contemptuous reception that system met with from many of the ablest senior surgeons of the time, men moreover who were perfectly sincere and honest in their disbelief, who were free from jealousy or spite towards those who introduced the new principle. They simply and honestly believed there was nothing in the proposal; some of them even supposed that after giving it a fair trial they had found it valueless. There were at that time juvenile critics, I must confess, who while honouring and respecting their seniors, were of opinion that certain of them had never fully grasped either the principle or the details of the method.

This experience was to the writer a forcible lesson as to the importance of preserving an open mind.

A new and valuable fact sometimes lies very near to us without our perceiving it. We had Salix in our materia medica and no lack of cases of rheumatism needing it for centuries before Maclagan and Stricker pointed out the relation of the one to the other; even when it was pointed out many of us refused to accept the precious new truth.

What I am anxious to do is to show grounds for the belief that something may be done in the direction

of prevention while no mischief yet exists, and also of arrest when the evil is already in progress; to show that the despair with which most of us regard rheumatic valvulitis is not well founded. That it is quite worth while to make energetic and determined efforts in every case we meet with to combat this most formidable danger.

Circumstances which led to the enquiry.

Perhaps some apology is due to the reader for the references to personal experiences which in this monograph are inflicted upon him. They appear almost inevitable. It seems desirable that the author should explain how he came to undertake the enquiry to which this volume refers, and that he also make some reference to the successive stages of the enquiry. As a student he fully imbibed the opinion of his teachers, that incipient endocarditis was of necessity to be left to fate, that no other course was possible. This was indeed the author's practice alike in hospital and in private for more than twelve years. Cases of acute rheumatism, sometimes with hearts sound at the outset, were treated; frequently they developed cardiac *bruits*; they always recovered from the rheumatism, but too often passed from under one's hands with the *bruit* still existing; and when the future history of these persons was traced they were commonly found to have become the subjects of chronic valvular disease. A series of five patients who in succession came under the author's charge with sound hearts, developed acute rheumatic endocarditis and recovered from their rheumatism, but had to recommence the toils of an active

life as labouring men, four with mitral, and one with aortic regurgitation. It was impossible to have so painful an experience as this (not to speak of others almost equally sad) without being profoundly impressed. If this was a type of the result of expectant treatment surely it was time to try something else, nothing one could devise could be much worse. It was a literal fulfilment of the words of Prof. Rosenstein of Leyden[1] in his treatise on this disease: "Endocarditis is almost certain to result in chronic valvular disease."

I set to work to look up authorities, to read what Hope, Walsh, Stokes, Skoda, Friedreich, Fuller, Petit, Rosenstein, Sibson, Barclay, Jaksch, Budd, Bamberger, Latham, Ormerod, Graves, Corrigan, Bouilland, Trousseau, Fraenkel, Niemeyer, and later authors had written, and then commenced experimenting with one or other of the methods some few of these authorities had recommended as preventive measures. One idea specially impressed and encouraged me; Jaksch of Prague had written on the natural tendency the heart had towards the spontaneous cure of its diseased valves[2]. This the more impressed me from the fact that a patient with endocarditis in my wards at this time lost the *bruit* and all signs of valvular mischief quite spontaneously, while lying quietly in bed undergoing treatment for something else.

Without detailing the various suggestions of the writers above named, which would occupy space somewhat uselessly, I may add that I proceeded to treat some cases with poultices, others with dry or

[1] *Von Ziemssens Syst.* vol. VI. p. 91.
[2] *Prager Vierteljahrschrift*, 1860.

wet cupping, or with leeches applied to the precordia, or with large blisters, or with ice bags over the same region. These attempts were carried on for nearly four years whenever cases occurred, but unhappily the results were not encouraging. It seemed most difficult to devise any method which would influence the heart. Probably one error made was in undervaluing the influence of prolonged rest. At length a triple method was attempted, which soon gave more encouraging results, it combined (1) Prolonged rest, (2) Continued gentle stimulation of skin surfaces related to the heart, (3) The use of certain absorbent and alterative drugs.

I don't know whether anyone has carried out for a long term of years a definite experimental scheme of treatment of cases of valvulitis, and published the results, if so I have not chanced to meet with the monograph. I determined to attempt a prolonged enquiry of this kind, and to record statistics of the results in so far as they could be obtained. Many years are required for such an experiment, for the cases needed are not very abundant. In fact the whole enquiry has covered the thirty years elapsing since I entered upon practice and became a hospital physician down to the present time. During the whole of this period I have been favourably situated for seeing cases.

I am far from saying that the method which nineteen years of observation and experiment have caused me to adopt is the best or the only means, or that it is a perfect method. All that can be affirmed is that after searching in more or less blindness and ignorance for some solution of the problem,

after various errors and failures, a method has been arrived at which is followed by far better results than those previously attained.

Thirty years' experience of the treatment of these cases has enabled me to contrast the results following, upon the one hand what may be termed the expectant method, with those secured on the other by the system to be described below. The difference is so overwhelmingly great and to my own mind so conclusive, that whether my professional brethren will believe me and try the method provisionally, or not, I must at any rate lay it before them.

If careful measures of prevention were carried out by the profession in general, I cannot but believe that a material lessening of the amount of heart-disease in our population and thus a great diminution of suffering would ensue.

CHAPTER II.

PATHOLOGY OF ENDOCARDITIS.

It is important to have a clear idea of the disease we are dealing with. What is the exact morbid condition which exists in the heart when the organ is attacked by rheumatic disease? We know that the muscular substance, the external fibrous envelope and the internal lining membrane, are all liable to be affected[1], and the last named, the endocardium, is of the three that most commonly attacked. Why the disease should assail the lining membrane of the heart rather than that of the vessels it is not very difficult to understand, nor is it hard to comprehend why certain parts of the endocardium are specially selected by the rheumatic process. The surfaces most markedly affected are the curved lines on the auricular surface of each mitral cusp, along which the two cusps come into contact when the valve closes, and the corresponding line in the aortic cusps (here on the ventricular side). The physical conditions of the momentary junction of the two mitral cusps are very peculiar, and worthy of a moment's study. At the instant of systole pressure rises in the left ventricle rapidly until it reaches an amount equivalent to that

[1] See Letulle, *Anat. Pathologique*, 1897, p. 98.

of a column of mercury between 200 and 250 mm. in height; it remains at that level for about ·08 of a second, falls for about ·2 of a second gradually and then rapidly. The two valve borders therefore have what may be described as a sharp and rapid contact for ·08 of a second under a very high pressure; the impact is so rapid and so acute as to bear perhaps as much resemblance to the stroke made by the point of an actively wielded whip-lash, as to anything else one can liken it to. This rapid and forcible contact is made and broken sixty or eighty times per minute, and during the intervals the blood torrent rushes over the surfaces in question at varying speeds and varying pressures. This small area on each valve is therefore subject to mechanical conditions of a specially arduous and trying kind[1]. It is in fact marvellous that an endothelial surface, even when perfectly healthy, should be able to withstand so severe a strain, more particularly when we remember that the tenuity of the cusp only permits of nutrition and repair through the agency of connective tissue channels, no blood vessels existing nearer than the attached margins of the cusp. Understanding these facts one can comprehend why the region in question should give way before others under the stress of a rheumatic attack. It may be that micrococci are actually "hammered" into the spaces between the endothelial plates. In some way the endothelia are destroyed; they are not quickly replaced; inflammatory products are effused and projected in bead-like masses or vegetations from the edge of the cusp, while the loss of endothelia constantly determines coagulation, and small

[1] See also Sibson in *Russell Reynolds' System*, IV. 458.

thrombi form on the damaged surface. Leucocytes are sometimes seen in the deeper layers of the vegetations, as almost constantly are masses of micrococci[1].

Although the crescentic area just referred to is the especial seat of rheumatic attack, the whole surface of the valve and the line of its attachments to the cardiac wall are liable to be affected.

As the endocardial mischief progresses the cusp tends to thicken, and in place of its normal, strong, tough, but pliable fibrous substance, there develope cellular elements in too great number, and the membrane becomes soft but yet less freely mobile. At the attached margin vascularity increases, leucocytes are effused, and the subendothelial layer thickens.

In the author's experience the mitral valve is affected at least ten times as frequently as the aortic. A primary affection of the latter has in fact been surprisingly rare in his clinique. Usually when the aortic has been affected it has followed endocarditis of the mitral; one imagined the rheumatic inflammation travelling from the mitral margin upwards to the base of the aortic cusps and spreading onwards to their free edges.

Cases of acute simple endocarditis are rarely fatal, in this respect they differ widely from the infective type which usually terminates at an early date in death. (With this latter form we of course have nothing to do in the present volume.)

During the whole of the writer's experience only two fatal cases have occurred in his practice. One was an extremely severe case of chorea in a youth, proving fatal in the acute stage of the mitral valvulitis. The

[1] See Ziegler's *Lehrbuch für Patholog. Anatomie*, p. 27.

cusps were studded with large white vegetations along the crescentic margins on the auricular aspect. This happened upwards of twenty years ago; the preparation was not kept. The second occurred lately; the patient entered the hospital moribund from another disease, while in the later stage of aortic valvulitis.

The author's friend and colleague Dr Hill Abram, Assistant Physician to the Royal Infirmary and Lecturer in the Thompson-Yates pathological laboratory of University College, has kindly examined this specimen and two others which he procured elsewhere. I am indebted to him for the plates annexed, and for the following pathological note in reference to them.

Note on the Morbid Histology of Acute Endocarditis (*Valvulitis*). By J. Hill Abram, M.D. (Lond.), M.R.C.P.

The morbid histology of acute non-ulcerative endocarditis has been but little studied, few observers being able to obtain material for investigation. The reason is not far to seek, for it is but rarely that patients suffering from the affection die in the early stage.

No apology is necessary therefore for recording the results of the examination of three cases, one of which, the most important, I owe to the kindness of Dr Nathan Raw.

A survey of the literature of the normal anatomy of the valves, as of the morbid histology, shows I think the scantiness of our knowledge. With regard to the special point I wish to discuss, the descriptions of

acute endocarditis (valvulitis) given in the various books on medicine seem to be simply a special application of the generally accepted views of inflammation, and take no notice of the fact that the valves in the main are devoid of vessels.

So far as my specimens go they support the views of O. Veraguth[1], and these are of sufficient importance to be given in some detail.

Veraguth describes the structure of the normal valves (auriculo-ventricular) as follows, excluding the endothelial layers and working from the auricular surface: (1) A thin layer of cells with slender elongated nuclei with a small quantity of protoplasm; (2) A broader layer with fewer cells and a considerable intercellular substance, the nuclei of the cells are large, round and granular. The intercellular substance is finely fibrillated; (3) A fibrillated zone with but few nuclei, thickened by the insertions of the chordæ tendineæ; (4) and (5) resemble (2) and (1), but are not so broad.

In the case of the semilunar valves the central fibrillated zone is wanting towards the free margin, the second and fourth layers therefore mingling.

The usual tests for the duration of an endocarditis are two, first, the degree of adhesion of the vegetation, and second, the character of the thrombus. The use of the first test tends to destroy the specimen, and the second is useless, for nothing is of more frequent occurrence than a recent thrombus on a valve with chronic disease.

Veraguth rightly, I think, says the decision in large measure must rest on the histological exami-

[1] *Virchow's Archiv*, Band 139.

nation. Briefly stated, in Veraguth's opinion acute simple endocarditis is characterized by an overgrowth of the second layer, *i.e.*, the layer containing cells with large nuclei. He further states that in the earlier stages there is no infiltration of leucocytes and no vascularization.

This is of interest as it shows a reversion to Virchow's view of inflammation, as broadly opposed to the vascular theory of Cohnheim now generally accepted.

The most important specimen I have examined was given me by Dr Nathan Raw. It was obtained from a rheumatic fever case, and the duration of the valvulitis was estimated to be three weeks. The valve affected was the aortic; the cusps on the ventricular surface, at some little distance from the free margin, were studded with small white vegetations, the cusps generally being thin and flexible.

A section examined with a low power, see Fig. 1, shows a vegetation AA on the ventricular surface of the cusp, capped by a small thrombus B; on either side of the nodule the cusp is apparently but little altered. The change is seen to involve the subendothelial layers on the ventricular surface, those on the aortic surface being but little affected. This distribution is better shown in the enlarged view of the vegetation given in Fig. 2, which also shows well the general nature of the new tissue at C, indicating the fact that we are dealing with a hyperplasia and not with a leucocytic infiltration.

Fig. 3 shows under a still higher power the margin of the vegetation D with a portion of the cusp E. In the vegetation the round large nuclei are well seen, and

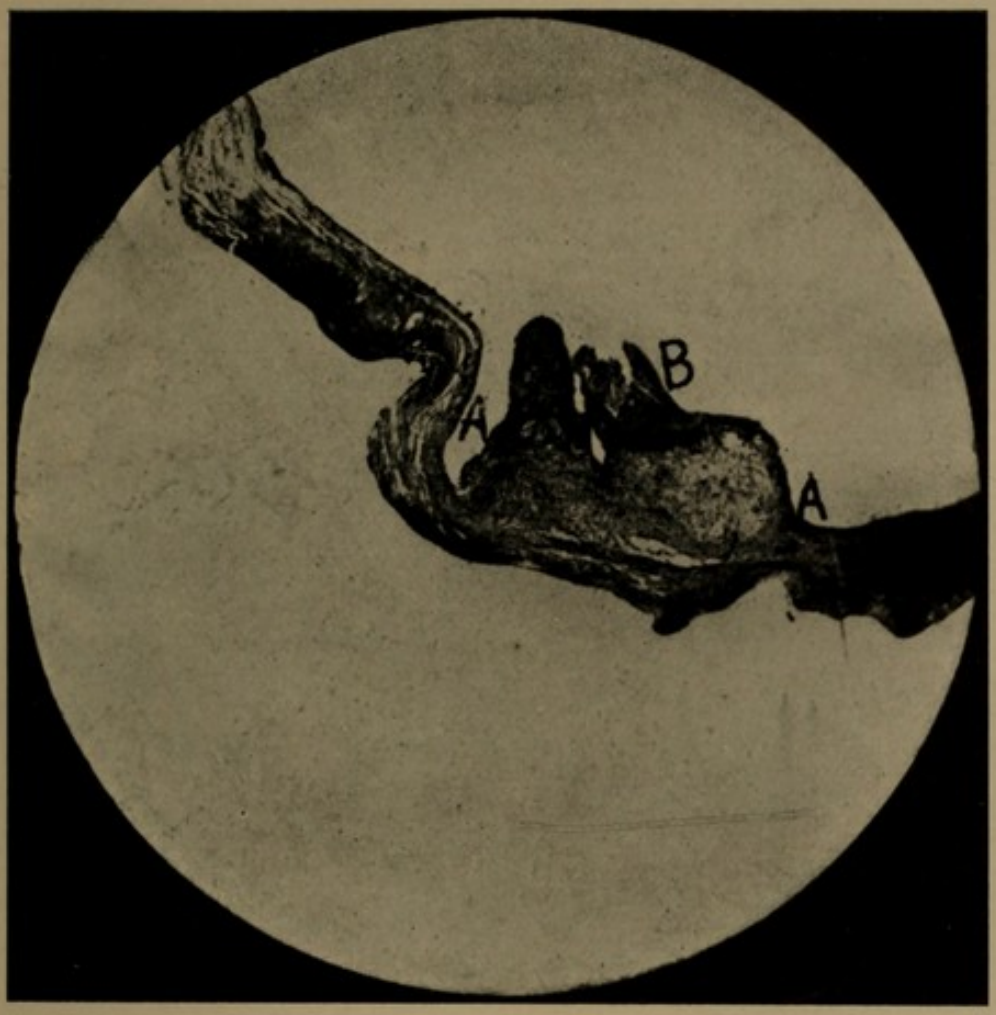

PLATE 1. SECTION THROUGH A CUSP OF THE AORTIC VALVE SHOWING RESULTS OF RHEUMATIC ENDOCARDITIS. (× 40.)

The free edge of the cusp projects a little to the right of the picture. *AA* is the diseased part of the valve, the section is cut through a vegetation on the ventricular aspect of the cusp. On the summit of the vegetation is seen a thrombus *B*.

PLATE 2. ENLARGED VIEW OF SECTION THROUGH THE VEGETATION IN PLATE 1. (×200.)

At *C* the hyperplasia of the fibrous tissue of the cusp is seen with considerable distinctness. At *N* a necrosis is occurring, causing a lack of clearness in detail.

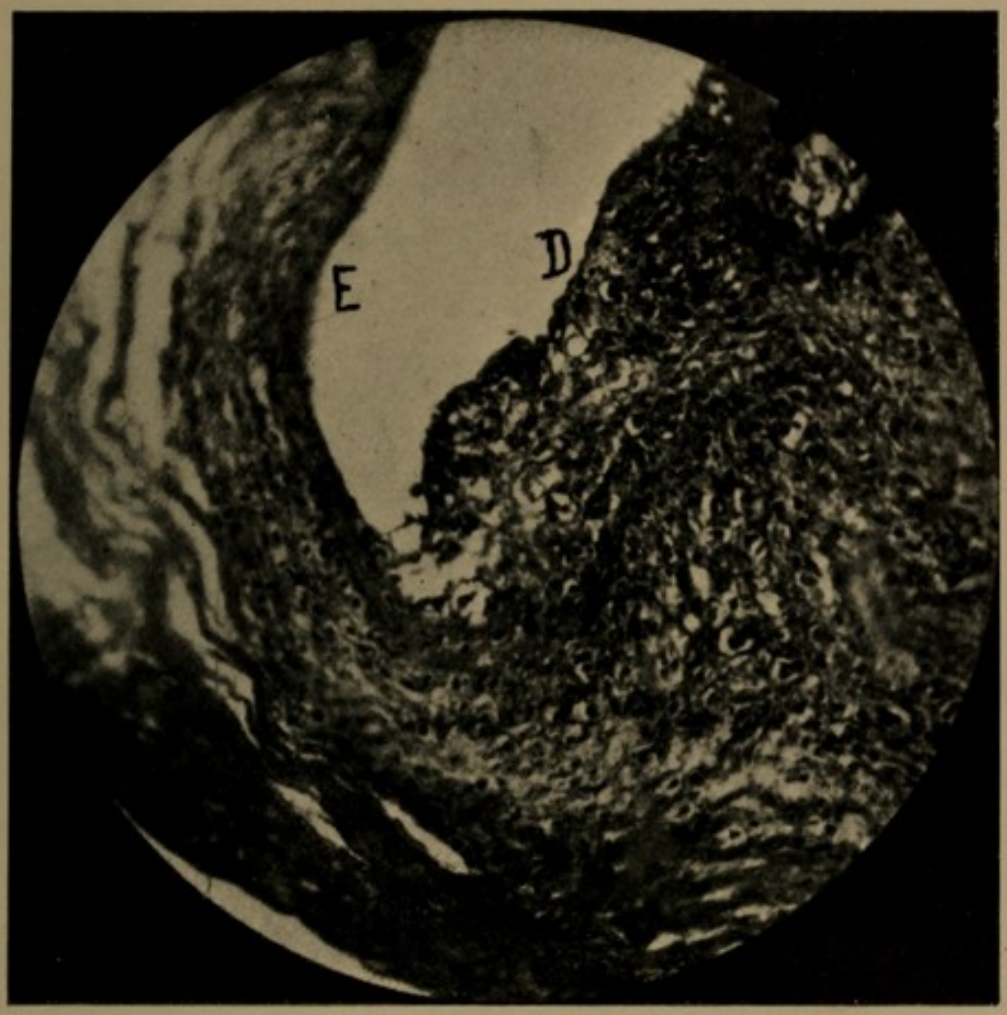

PLATE 3. SECTION STILL FURTHER MAGNIFIED OF THE LEFT END OF THE VEGETATION SHOWN IN PLATES 1 AND 2. (×500.)

D is the left-hand edge of the vegetation. The large round nuclei are seen with some distinctness and may be contrasted with the smaller and more elongated nuclei of the adjacent part of the cusp *E*, in which little or no hyperplasia has occurred.

contrast with the smaller, more elongated nuclei of the subendothelial tissue of the unaffected portion of the cusp.

Fig. 4 is taken from the base of the thrombus. The nature of the cells at F is not quite plain, they may be and to my mind are proliferating connective tissue cells, but are possibly leucocytes.

In none of my sections, nor in the photographs is there any leucocytic infiltration, or evidence of vascularization.

Fig. 5 represents a section from a rheumatic case in which there was no clinical evidence of septic disease, nor at the post mortem was any found.

In this case the vegetations were large and very friable and covered the entire cusp. The photograph was taken from an area near the base of the cusp. The fibrillated core G almost devoid of nuclei is shown, clothed on either surface with an amorphous necrotic tissue H, representing the subendothelial tissues in a state of coagulation necrosis. Here too leucocytes and vessels are not to be found.

This specimen is important, for it is obvious that with such marked necrotic change little result can be expected from any line of treatment.

The letter N in Figures 2 and 4 indicates a patch of necrosis in this less severe type of endocarditis.

My third case was identical with that from which Fig. 5 was taken, and therefore no photograph is given.

Fig. 6 is a low power photograph of a chronic aortic valvulitis. As in the first case the main changes are seen in the ventricular subendothelial layers K which now consist of a dense well-nucleated fibrous tissue,

the dark portion L is slightly calcareous. The other interesting feature is the densely nucleated central layer probably containing vascular spaces M.

In all three cases numerous masses of staphylococci were found in the superficial parts of the diseased tissue. There was no evidence of ulcerative disease in any one of the three cases examined.

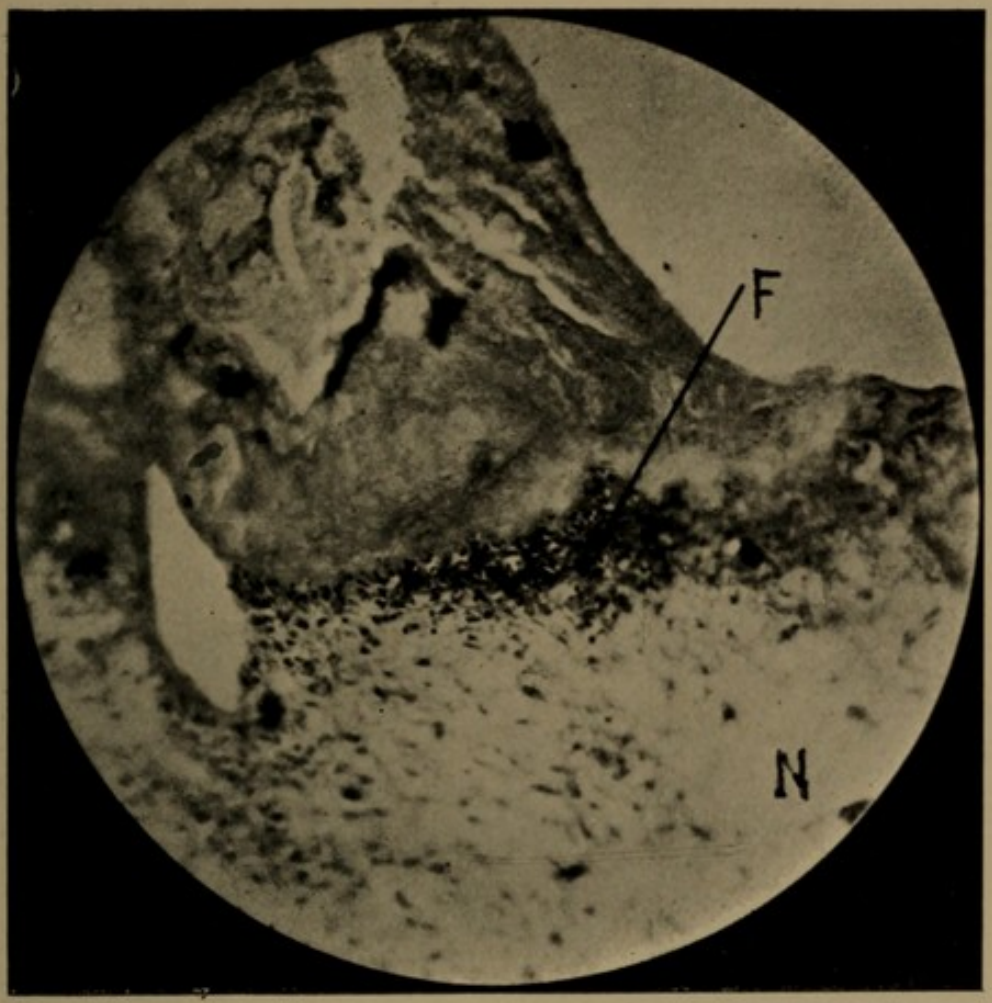

PLATE 4. HIGHLY MAGNIFIED SECTION THROUGH THE THROMBUS SHOWN IN PLATES 1 AND 2. (×500.)

F is the area at which denudation of endothelia has occurred and on which coagulation and the formation of the thrombus has taken place. At the line of attachment of the thrombus a number of small but distinctly marked cells are seen at *F*; they are probably proliferating connective tissue cells, but may possibly be leucocytes. At *N* necrosis is occurring.

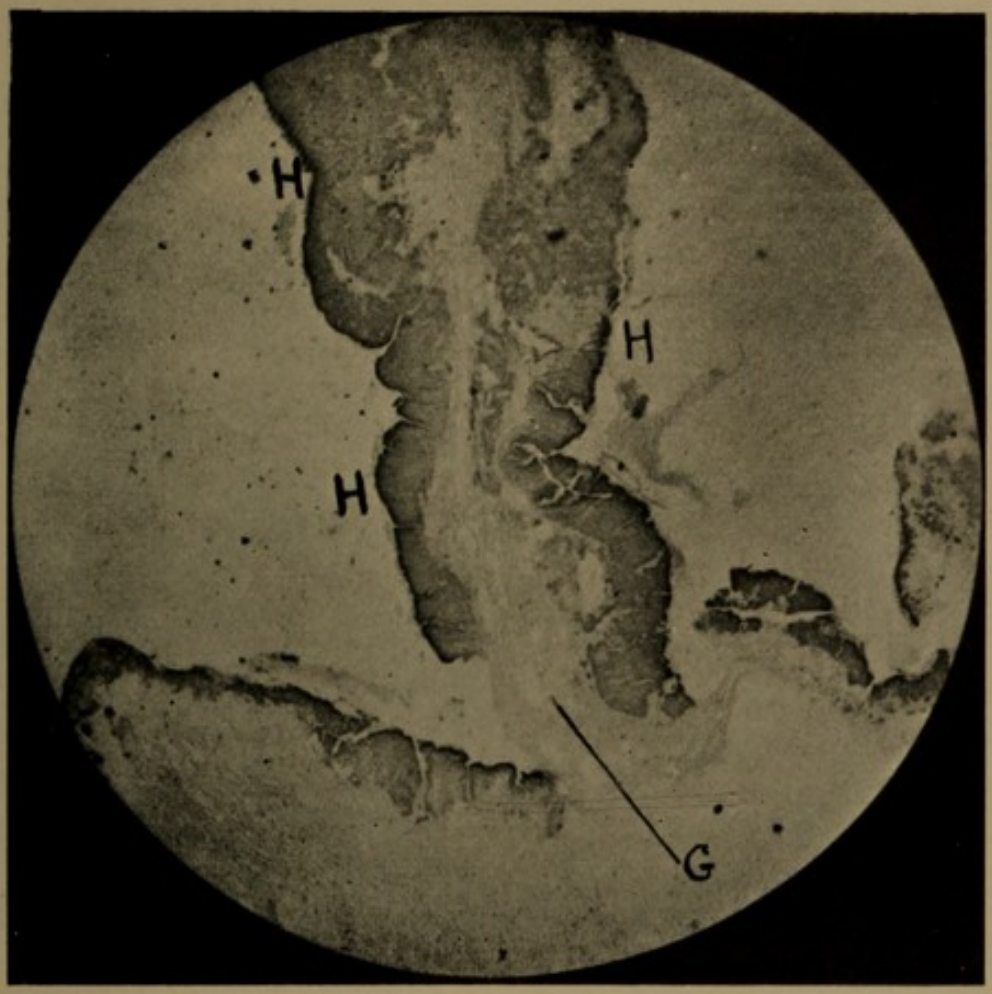

PLATE 5. SECTION THROUGH AN AORTIC VALVE IN A LATER STAGE OF RHEUMATIC ENDOCARDITIS. (×200.)

Large and friable vegetations *HH* in a state of coagulative necrosis are seen covering the cusp on both aspects. The fibrillated core *G* is seen to be almost free from nuclei.

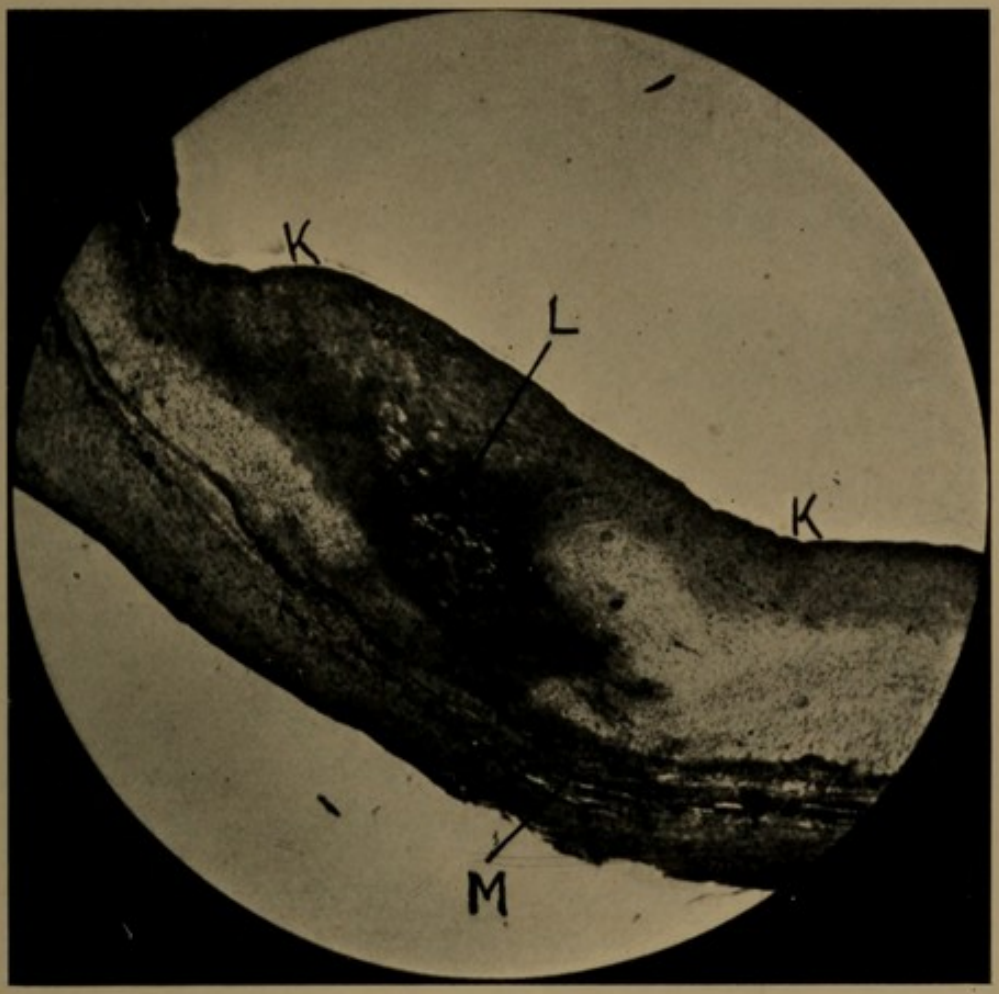

PLATE 6. SECTION THROUGH ANOTHER AORTIC VALVE IN A LATE STAGE OF RHEUMATIC VALVULITIS. (×55.)

The cusp is seen to be quadrupled in thickness. The layer (nearly an inch broad) below *KK* consists of the dense, highly nucleated fibrous tissue of the subendothelial layer on the ventricular side. Calcareous degeneration exists at *L*. The densely nucleated central layer at *M* seems to contain vascular spaces.

CHAPTER III.

PHYSICAL SIGNS OF ENDOCARDITIS.

ALTHOUGH so great an amount of time and labour has been devoted to the study of the physical signs indicative of heart-disease, that method of diagnosis has not lost all its difficulties and uncertainties. Among the fields of enquiry which still remain obscure must be included the earlier stages of valvulitis. We can scarcely be said to know of any signs which give warning or evidence of the earliest stages; sometimes there is a vague sense of discomfort about the precordia, or a rise of temperature otherwise unaccounted for, or an acceleration of the heart's action. More frequently none of these occur.

The first distinct physical sign which indicates, at a somewhat later stage, that something is amiss is a softening and dulling, and sometimes a prolongation, of the sound at the apex caused chiefly by the systolic closure. Normally, the systolic sound at the apex is compounded of two elements, (1) the muscle sound produced by the complex though non-tetanic contraction of the ventricle—this the smaller factor—and (2) the "crack" of the distended valve cusps, suddenly made "taut" as the blood-stream with great force fills and strains them; this I think is much the more potent of the two factors.

In commencing mitral endocarditis, this sound is exchanged for a longer and duller tone, due most likely to the muscle sound and to a less complete and probably slower valve closure. The valve membrane is becoming stiffer, less pliable, and does not so immediately adapt itself to the blood current. The sound becomes more prolonged, gradually assumes a "whiffing" character, due as one supposes to the partial escape of fluid between the thickened and imperfectly mobile cusps, which do not allow of close and accurate apposition of their edges. It is easy to understand that the thrombi and the projecting vegetations which form on the edge of the cusp (as shown in plate 1 A and B) are extremely likely, or indeed certain, to prevent exact closure. Hence the whiffing or faint whistling sound produced. This sound (which of course varies greatly in different cases) soon changes into a distinct regurgitant *bruit* as the change in the cusps becomes so far advanced as to compel a considerable reflux at every systole. This large reflux at every contraction raises pressure in the pulmonary veins, in the capillary system, and in the arteries of the lung, with the result of at length so augmenting pressure in the pulmonary artery proper as to cause a sharper and louder closure of its semilunar valves—a closure often louder and more accented than that of the aortic valve. The apex systolic *bruit* is now commonly heard in the axilla, if loud enough.

As this process advances in some cases the cardiac dulness slightly increases, the sign I imagine of dilatation or of myocarditis; in more instances, judging from my own experience, it remains unchanged; in a

few again not only do myocarditis and dilatation occur, but the pericardium is involved and fluid collects.

It may here be remarked that although we note the onset of a *bruit* with apprehension, as an indication of endocardial mischief, I personally also feel that, given the fact of active endocarditis, there are in the meantime certain advantages secured by the existence of the regurgitation, as follows—(1) regurgitation lowers pressure within the ventricle, thus interfering less with the reparative process than would be the case if the pressure were normal; (2) the lowering of pressure on the arterial side, and the relative increase on the venous, cause the patient to feel weak and disinclined for exertion, disposed rather for the recumbent posture, thus establishing conditions more favourable for arrest of the mischief and for repair of the injured valve.

The position and arrangement of the thrombi and projecting vegetations on the edge of the cusp, also of the contractures which may occur later, vary widely, with of course equally varying results, not only as to the amount of reflux or of stenosis, but also in the character of the sounds produced.

Sometimes, though rarely, regurgitation occurs without giving rise to a *bruit* of any kind. In my experience, however, these cases are very unusual. Great variations undoubtedly occur in the quality and loudness of the *bruits* which accompany rheumatic valvulitis.

Diagnosis of mitral or aortic valvulitis.

How are we to determine whether or not valvular mischief is impending? How to distinguish between

the *bruits* of anæmia or of recent or old standing dilatation or those caused by myocarditis, on the one hand, and those of commencing valvulitis on the other?

(1) Hæmic *bruits* are not difficult of detection, the anæmia of the patient helps us, also the position of the murmur, its focus being usually at the pulmonary valve; the accompanying cephalic *bruit* or the venous hum in the neck make diagnosis easy. Of course an endocarditis may readily occur in an anæmic subject, and a regurgitant *bruit* at the apex or base co-exist with a hæmic murmur at the pulmonary valve.

(2) The *bruit* of acute dilatation is much rarer. The condition sometimes occurs without any *bruit*. In my experience acute dilatation along with rheumatism is much commoner in text-books and monographs than at the bedside.

The common chronic cases of dilatation are not likely to be mistaken for acute valvulitis.

(3) The *bruit* of relative or muscular incompetence due to myocarditis of which some writers speak, is in my opinion not very common apart from endocardial trouble. It may form a factor in the production of the *bruit*, and if so it is very difficult to distinguish how much is due to it and how much to the valvulitis. The *bruit* we commonly meet with occurs early in the course of the disease; doubtless Dr Sansom is right in pointing out that the apex murmur occurring early in acute rheumatism is not due to febrile change in the muscle, but to genuine valvulitis[1].

[1] *Lettsomian Lectures*, p. 18.

CHAPTER IV.

TREATMENT OF ACUTE RHEUMATISM WITH A VIEW TO THE PREVENTION OF VALVULITIS.

Before describing the measures employed to secure the arrest of commencing cardiac mischief, it may be well to say a few words about the treatment of acute rheumatism and the efforts made to prevent any valvular complication.

Being of opinion that during the acute stage chills are especially hurtful from their tendency to cause extension of the rheumatic inflammation to other joints and to the viscera, I always have each patient clothed in a long, warm flannel night-dress, of which garments a large stock is kept in my wards. This is better than wearing a cotton night-dress and lying in the blankets.

The profuse sweats which occur in the complaint are apt to occasion unpleasant and injurious chills unless this precaution is taken. It has always appeared to me that the salicylates act better and produce less unpleasant effect if a cholagogue be given at the outset and be occasionally repeated during their administration; accordingly a moderate dose of pil. rhei co., fortified if necessary with a little calomel or podophyllin, is administered and repeated as may be

found necessary. Salicylates are of course at once commenced in full dose, the amount and frequency being regulated by the nature of the case. Any specially painful joints are wrapped in cotton wadding, the softness and support of which seem to lessen the pain and discomfort. Absolute rest in bed is of course enjoined. In some cases alkalis are prescribed along with the salicylates. A light diet consisting chiefly of milk is given. If the pain and sleeplessness demand it a small dose of morphia is ordered for the first night, it is rarely needed a second time. As a rule the pain and fever are much reduced within the first twenty-four hours. In case the pain remains obstinately in any joint I always use small blisters adjacent to the part, discs about the size of a florin are quite large enough, they occasion practically no pain or discomfort, if properly attended to, and speedily remove all pain. I personally have been fortunate in rarely meeting with cases of rheumatic hyperpyrexia. If such should occur, we maintain the flannel covering over the thorax and limbs, but apply cold compresses to the abdomen, employing ice if needful.

It is scarcely necessary to say that the heart is carefully examined on admission and daily afterwards. If a *bruit* is heard when the patient is first seen, efforts are made to determine whether it be recent or of long standing, by examination of dulness, impulse, &c., and by enquiries as to duration of symptoms, previous attacks and antecedent signs of cardiac disease.

If there be clear evidence that valvular disease of long standing exists, no further treatment of the heart is usually attempted. In some instances it is difficult to determine this question.

In about three days' time the patient frequently says he is quite well and expresses a desire to get up. The author being strongly of opinion that treatment by salicylates is to be regarded rather as a valuable mode of repressing the pain and fever than as a means whereby the disease is quickly put an end to, believes that however well the patient is, he should remain in bed at least a fortnight or three weeks. If this is not done, and if the administration of salicylates is not maintained during this period in small doses, relapses are very likely to occur, and in such relapse there is special danger of the onset of endocardial mischief. In the author's opinion the strict enforcement of this rule and the protection afforded by the use of the flannel garment named, tend to early recovery, freedom from relapse, and exemption from endocarditis. Among several hundreds of cases treated, the percentage of endocarditis has been exceedingly small; even including the large number which came into hospital with signs of disease, the proportion of cases is only about seventeen per cent.

So much for efforts at prevention.

CHAPTER V.

METHODS EMPLOYED FOR THE ARREST OF COMMENCING ENDOCARDITIS.

However diligently the physician may use all means within his control to prevent endocardial mischief, some cases are sure to occur in spite of his efforts. More frequently will he meet with cases in which the disorder has commenced before the patient came under his charge. What means can be adopted to arrest the advancing mischief, to prevent it proceeding to the point of acute inflammatory change with contracture, deformity, adhesion or necrosis, the shortening of the chordæ tendineæ, or their destruction by ulceration?

Personally the author has no doubt that the lesser degrees of morbid change are susceptible of remedy, while the more advanced ones are beyond it. It is doubtful if even the condition represented in Plate 1 above has not advanced too far for repair.

When once necrosis occurs, as is shown in Plate 5 H, all hope of return to a healthy state is obviously at an end. So also when chronic thickening has taken place and the cusp is tripled or quadrupled in depth of tissue, and patches of calcified material form in its substance, as seen in Plate 6 L, there can be little hope of return to the physiological condition.

The question of the speed with which these changes occur is an interesting one. Can we state any limit of time beyond which all hope of amendment is vain? In the author's opinion no very definite limit can be given, because the acuteness of the destructive process varies in different instances. In general however the writer's experience has caused him to believe that the chance of restoration is small if more than three or four weeks have passed without due care and precaution being taken. And particularly is this the case with adults; in children and youthful patients it would appear as though the period during which repair may occur is on the average more prolonged.

If careful treatment is commenced at once and persevered in sufficiently long, in the great majority of early cases restoration takes place.

What are the methods by which we may hope to arrest the destructive changes which are being wrought in the heart and to favour a return to a more normal condition?

They are three in number, in so far as the present writer's method is concerned.

(1) *Treatment by rest.*

The first and not the least important is Rest. Probably everyone will admit the probability of benefit from this condition.

In diseased states of any of the tissues or organs of the body we seek to secure for the ailing part as much rest as is possible. It seems indeed to be difficult for full functional activity and a reparative process to co-exist. In many forms of disease it is practicable to

reduce or to arrest function for a time. The complete restoration of an acutely rheumatic joint which we so constantly see, probably would not be effected, or only be effected with difficulty, if the joint were in constant movement. Stern but kindly Nature gives us the warning of pain and thus secures the needful rest. No complete repose is possible for the toiling heart; whatever its own condition may be the duty owed to the organism cannot be intermitted for a moment. Not improbably the cause of the rarity of complete recovery in the heart, when left without special treatment during acute rheumatism, is this lack of rest. Consequently in treating a case of early valvulitis we are anxious to restrain, in a measure, the force and velocity of the blood current, to lessen, as far as it can be lessened, the work of the heart, to diminish the dangerous pressure of the blood wave on the weakened and softened valve cusps, to lengthen as far as is practicable the brief rest-periods of the heart, between its systoles. To secure in fact the most complete physiological rest, alike of mind and body, reducing the demands made upon the heart to the physiological minimum. No muscle work is permitted excepting that involved in such respiratory and circulatory effort as life requires; all stimulating food is withheld and only a light, simple, easily digested diet is given; of course the patient is kept in the strictest recumbency, no raising of the head is allowed; no getting out of bed for any purpose; and everything tending towards mental agitation or disturbance is carefully avoided. Pain is prevented if possible, and with this object, the speedy relief given by the salicyl compounds to the rheumatic patient is of the greatest

value. If pain arises from any other cause it should be combated and a large amount of sleep secured.

The maintenance of strict physiological rest is essential for several weeks. As a general rule the patient is quite willing to resign himself to it. If he is not, it becomes the physician's duty cautiously to explain to him the reasons which render repose imperative, avoiding as far as he can any phrase which would excite fear or alarm.

This first measure of treatment, rest, is perhaps the most essential of all, and though I believe the others to be yet described are also of importance, no doubt some cases recover which are treated by strict rest alone.

(2) *Treatment by stimulation of trophic centres.*

It is difficult to discover a channel through which one can directly influence the heart, so as to stimulate its reparative processes, without either impairing vitality or augmenting functional activity. I strongly believe that those drugs which act directly and specifically on the heart, the so-called cardiac tonics, including digitalis and caffeine, are distinctly injurious if administered during the existence of valvulitis. Setting these aside does any other channel present itself?

Can we get any hint from methods of treatment which influence rheumatism favourably in other regions?

Next in value to the salicylates in the treatment of articular rheumatism comes in my belief the employment of small local blisters. Those who have witnessed the effect of Dr Herbert Davies' method of treating

Rheumatic Fever[1] by the application of small blisters adjacent to the joints, know how great was its efficacy. Personally I have always used small blisters when a rheumatic joint proved refractory, and rarely without good result.

After employing this method in hundreds of cases (including my own person), there are few things about which I have less doubt, than in regard to the speedy and complete relief afforded, and also as to the trivial nature of the pain caused by the blister—if the latter be properly managed. It is rarely needful to employ a blister larger than the size of a florin. A small poultice should be applied after it.

What is the explanation of the beneficial action of this so-called counter irritation? Why do pain and swelling disappear so rapidly from a rheumatic joint if we apply one or two small discs of canthos or other form of vesicant to the skin adjacent to it?

The irritation of the sensory nerves of the skin probably causes excitant impulses to traverse the trophic and vaso-motor nerves of the part, whereby dilated arterioles are contracted and increased activity of the natural reparative process is promoted. Is there any other reasonable theory accounting for this well-established fact? If there is I have yet to hear it.

This so-called "counter irritation" is I believe in reality direct stimulation of the vaso-motor and trophic nerves of an ailing part. It is possible to stimulate not only the nervous system as a whole but individual parts of it. If a cicatrising wound or a callous ulcer is slow and sluggish in its progress do we not stimulate

[1] *Clin. Lect. and Reports of Lond. Hospital*, 1864.

its nerve filaments by the application of some form of local excitant? if in the late stage of a conjunctivitis the vessels remain dilated, does not the application of a solution of silver nitrate speedily reduce them to their normal calibre? So in many other familiar forms we have the principle exemplified; by artificial stimulation we excite the local reparative powers to an activity they would not otherwise manifest.

A securely founded belief in the efficacy of the small blister treatment of rheumatic joints and in the theory accounting for it, suggested to me the question —If stimulation of the surface skin will so potently assist recovery in a rheumatic joint, is it not possible that the same beneficent change might also occur in the rheumatic heart if continued stimulation of its related skin areas were practised?

The upper, middle, and lower cardiac nerves on each side, as everyone knows, are associated with the three cervical ganglia of the sympathetic and through them probably with the upper eight dorsal intercostal nerves. Through the upper and middle cardiac nerves (which also communicate with the pneumogastric) the heart appears to be much more closely in relation with the first four dorsal intercostal nerves than with the remainder.

The researches of Head[1], Sherrington, Gaskell and others have made us acquainted with various regions of the surface skin which appear to be specially related to individual viscera within the body-cavity, and with none more definitely than that which coincides with the distribution of the 1st, 2nd, 3rd, and 4th Dorsal nerves and is associated in the closest manner with the

[1] *Brain*, 1893—1895.

nerve mechanism of the heart. We all know how frequently in angina and overstrain of the heart as well as in other morbid conditions of that organ, pain is referred to the suprascapular region, to the area between the clavicle and the nipple and to the ulnar side of the arms. This cutaneous surface and its nerves being so intimately related to the heart, it appeared quite probable that impulses might be transmitted by the afferent neuron *viâ* the cord and the sympathetic ganglia direct to the cardiac trophic nerve centres, and it was hoped that in the rheumatic heart results might follow stimulation similar to those which occur in a blistered rheumatic joint. At first sight the greater distance intervening between the thoracic skin and the heart seemed an objection; but just as ten yards or a thousand are practically equal in conduction along a telegraph wire, so an extra foot of transit would make little difference to a nerve impulse coursing along the sensory neuron, provided the route were a definitely co-ordinated one. At an early stage in the enquiry I had seen evidence from galvanometrical experiment that stimulation of the surface skin modified the electrical potential in the muscles of organs within the body-cavity, also that similar stimulation caused changes in the calibre of arterioles in those organs. Vaso-motor and electrical functions being influenced by skin stimulation it seemed not unreasonable to assume the same in the case of trophic function. There appeared to be a certain amount of antecedent probability that such a channel of nerve influence existed and was available for therapeutic purposes. Of course it must be admitted that the vagueness as to evidence, which is and always has

been the reproach of therapeutics, in some degree throws a shadow of doubt over this form of treatment. It seems impossible to offer conclusive proof in any given instance that the placing of a series of small blisters on the area named has definitely influenced the process of repair in a diseased valve. All I can assert is that such treatment is apparently followed by an increased steadiness and quietness in the action of the heart, and that when employed in a considerable number of cases of valvulitis, restoration of the normal action of the valve has resulted, in a strikingly large number of instances.

The method is reasonable, it has a scientific basis, founded partly on fact and partly on analogy, and the results attained are highly satisfactory.

The small blisters are best applied in front or in the axilla, the former by preference. If applied in the supra-scapular region they cause discomfort and interfere with rest. The region between the clavicle and the nipple on the right and left sides appears to be the most suitable.

(3) *Treatment by absorbent drugs.*

When a section of a cardiac valve affected by rheumatic inflammation is examined under the microscope it is obvious that a considerable amount of new and imperfectly organized material has been effused into the substance or upon the external surface of the cusp. If this effusion remains it frequently developes into permanent tissue; it may then undergo shrinkage or contraction; it may by its pressure interfere with the nutrition of the cusp and occasion a necrosis. If any of these changes occur recovery is

prevented. Therefore it is a matter of great importance that the effused material be absorbed and removed as quickly as possible. The two methods of treatment already named probably aid in the accomplishment of this purpose, but in addition it is desirable that such drugs as are believed to have an influence in helping to absorb effusions and to remove thickening in the fibrous tissues should be administered. Of these the iodides and mercury are the chief. Sodium iodide is perhaps in its other effects the least injurious of these, and accordingly I always administer it. In a few instances where the general health of the patient seems to warrant it, and when improvement in cardiac physical signs is tardy, small doses of calomel are given with care and in strict moderation.

These three methods therefore are adopted in conjunction. Critics have pointed out how much wiser it would be to experiment only with one of the three at a time, so as to find out if only one of them be the really efficient agent.

My reply is "vita brevis, ars longa." Another lifetime would be needed to experiment with these measures singly. This combination of means has yielded good results, there is fear of diminishing the success achieved if some of the means be omitted; if my own heart were assailed most certainly I should employ all the three measures, therefore I do so in the case of others.

CHAPTER VI.

PHYSICAL SIGNS INDICATING ARREST AND RECOVERY.

Much patience is required in the treatment of these cases. The time elapsing before the *bruit* disappears is variable. In the author's experience the general rule has been that the earlier careful treatment and strict rest are begun after endocardial symptoms are observed, the earlier does improvement manifest itself. If however relapses of the rheumatism occur the duration of the signs of valvulitis is apt to be extended. In general no sign of improvement is seen until two, three, or four weeks after all rheumatic pain and pyrexia have disappeared. It is during this period, when the patient feels well and has a natural desire to leave his bed and return to his former course of life, that the need for patience is so great. The first hopeful indication is a gradual lessening in the loudness of the *bruit*. Next, the *bruit* becomes to a considerable extent influenced by posture; when the patient is recumbent it will be heard, but when he sits up (which he is rarely allowed to do) it becomes almost or entirely inaudible. It becomes variable also, being heard at some times and

not at others; usually all this time the accentuation of the second pulmonary sound continues; at length the *bruit* disappears altogether, whatever the posture the patient may assume. For a considerable time rest and quiet are still necessary. In a few instances some signs of hypertrophy have been observed, also an impulse of somewhat peculiar character, having a partial resemblance to a thrill, has been noted when the patient first begins to go about. If a reasonable amount of rest is observed these signs usually disappear in a few weeks time. The accentuation of the second pulmonary sound also usually vanishes. It is especially important that care be taken to prevent a recurrence of rheumatism, for if in a case of apparently cured endocarditis another attack of rheumatism occurs, the valve is almost sure to be attacked again, and if so attacked the valvulitis resulting only yields to treatment after a very considerable lapse of time, in fact in many cases fails to yield at all.

In two or three cases in which the patient had failed entirely to lose the *bruit* in hospital and further rest was recommended at home, the patient on coming to be examined some months later was found to have lost the *bruit* and to be to all appearance well.

CHAPTER VII.

SUMMARY OF RESULTS.

At the date of going to press the scheme of treatment detailed above has been employed in eighty-six cases of endocarditis. I have of course met with a far larger number of cases than is represented by this number. Experience long ago made it obvious that when several weeks had elapsed since the onset of the malady little chance existed of complete restoration. When no measures are adopted, during the first two, three or four weeks after the onset of valvulitis, thickening, rigidity, and adhesions form, followed by necrosis or perhaps by the deposit of calcareous material within the cusp, or by shortening or ulceration of chordæ tendinæ. After that has occurred restoration is in all probability hopeless. It is indeed probable that even in these desperate circumstances, while recovery is not to be hoped for, nevertheless rest and other forms of treatment may restrict and limit the mischief which is in progress in the valve. I have consequently often employed the treatment in cases which I had no hope of curing. Cases of this class however I shall not speak of now. The eighty-six cases of which details follow belong to two categories.

1st. Those in which the endocardial mischief has come on under my own eye; the patient while under treatment in hospital for acute rheumatism was assailed by rheumatic valvulitis. These are highly satisfactory cases. Although it is true that the earliest working of the rheumatic disease in the valve is undiscoverable by any physical or other signs, so that in each of these cases the lesion was present some days before it was observed; still no great amount of mischief has as yet been done when the first signs of *bruit* become manifest. So often have I seen complete and permanent recovery take place after clear and unmistakable signs had existed of mitral reflux, that if I am sure of any thing in medicine I am sure of the possibility of this restoration.

The number of cases in which the disease came on while the patient was under observation is thirty-one. Of these twenty-eight made a good and, in all cases excepting two, to the best of my knowledge a permanent recovery. I cannot of course be certain that in no instance relapse took place later; every patient was informed of the risk he or she had encountered, warned as to future care, and advised to return if any further trouble occurred. Many came back again and again, and nearly all were found to have healthy hearts. Some were never heard of again, and all enquiries after them proved unavailing.

2nd. Those cases in which the valvular mischief had become manifest before the patient came under observation. These were the majority. The disease was probably recent, though not certainly so, whether recent enough to render complete cure possible was often doubtful. No distinct or long lasting history

of dyspnœa being given, and there being evidence of recent rheumatism only, there seemed considerable probability that the heart lesion was not beyond remedy. The number of these cases amounted to fifty-five. I had much less hope of course when in uncertainty as to the actual duration of the endocardial mischief, and anticipated failure in a considerable proportion. Out of the total number thirty-six left hospital with apparently normal hearts, while nineteen on dismissal had signs of more or less valvular disease. This however is not an unfavourable result. A considerable number of these cases have been kept under observation subsequently.

I give below brief notes of eighty-six cases, the favourable and the unfavourable alike. All were treated, in so far as the heart complication is concerned, in the same manner. Absolute rest in bed, the duration of which I was taught by experience to lengthen. In some cases, from unwillingness on the part of the patient to remain, from great pressure on our beds, or from other causes, this rest period was too brief. Small blisters were always applied to the upper part of the thoracic wall, viz., that between the clavicle and the nipple. In some instances they were placed below the nipple, but the higher position is probably the more effective, as being in closer physiological relation with the heart. Sodium or potassium iodide was always administered, excepting in the few instances in which it was deemed advisable to give mercury. The patient was always dressed from head to foot in flannel. The diet was light, chiefly consisting of milk.

In some instances the records of the cases are

meagre, and some of the details one desires are occasionally not to be found in the record. That I fear is not an uncommon experience in dealing with hospital records.

Some of the earlier cases are especially defective.

It is a matter of great regret that the subsequent history of many of the cases to be narrated was not followed for months or years subsequent to the apparent recovery from the cardiac attack. At first for some years the author had only faint hopes that he was really being successful; it was only when success seemed to follow success too frequently to be a matter of mere coincidence, that he really believed that the results were so much better than he had secured in former times. Thus for some years less trouble was taken to follow up cases. In many instances it has proved impossible to trace the patient. Sailors, servants, clerks, and labourers, disappear, letters are returned from the dead letter office, visits to the addresses they gave when in hospital result in failure. If the sick man has fully recovered he soon gets occupied with his daily work and does not find time to report himself at the hospital as he was requested to do. Provided he is conscious of being perfectly well he feels too little interest in his former illness to take the trouble to report himself merely because the doctor begged him to do so. Hence unfortunately the after history of many remains unknown.

It happens also most unfortunately that memoranda which were obtained in reference to the later condition of a few of the cases treated about ten years ago have been lost. The reports, made after careful examination of the heart, all showed a satis-

factory condition of the organ, but it is now quite impossible to say to which cases they referred.

The longest period during which a case of apparently arrested valvulitis has been kept under observation is in the instance of a private patient who lost the *bruit* and other signs of mischief more than fifteen years ago. He has been examined as regards heart sounds, dulness, physical vigour, and freedom from dyspnœa at intervals. When last seen some months ago his heart was absolutely normal. As I have in the following series of abstracts strictly confined myself to Hospital cases recorded by Registrar, House Physician or Clinical Clerks, this private case is not included.

CHAPTER VIII.

ABSTRACT OF CASES.

Case 1. John M. 26, carter, Admitted 17 Nov. 1884, Discharged 30 Jan. Case reported by House Physician and clerks.

No history of Rheumatism. Exposed to wet a fortnight ago, remained in wet clothing. Severe pain in nearly all joints. Temp. 102·2°. Sleepless. Delirious for many nights. Heart sounds normal. Small benefit from salicylates; deafness and enfeeblement of heart's action followed their administration, so on 29 Nov. the salicylates were stopped. Guaiacum and Potas. Iod. and alkalies given. Pain still severe. Complexion dusky, tendency to collapse: blisters applied to joints. 5th Dec.: Salicylates resumed with stimulants and morphia. 6th: Pain somewhat lessened. Colour improved, but pulse enfeebled. Dose of salicylates diminished. A systolic *bruit* at the apex manifest to-day. 9th: Mitral *bruit* strongly marked, pains still present, temperature falling. 11th: Pulse stronger. 12th: *Bruit* less distinct. 14th: Blisters applied to some joints and hot air baths ordered. 20th: Pain mitigated in joints. 29th: Pain now gone. Jan. 5th: *Bruit* distinct. Pain recommenced in the hand and neck, salicylates repeated. Small blisters to painful joints. Patient gradually recovered from the rheumatism. Repeated attacks of high temperature at intervals during November and December. He at length left hospital free from pain, but with marked mitral regurgitation.

This was an example of a class of cases one occasionally meets with in which no drug given internally appears to influence the rheumatic condition. The measures employed to remove the endocarditis were quite ineffective.

Case 2. Johanna M. 12, school girl, Ad. 15 June, 1885, Dis. 13 July. Case reported by H. P. and clerks.

History of rheumatism for three weeks. Pain in joints severe. Temp. 103°. Sweating profusely, much thirst, little sleep. Marked systolic *bruit* at apex. Vomiting.

Under treatment the pain gradually disappeared. The *bruit* became less marked and was entirely absent when she left hospital on the 13th July.

Case 3. George M. 18, ship's steward, Ad. 6 Jan. 1886, Dis. 23 Feb. Case reported by H. P. and clerks.

Had several previous attacks of rheumatism. Has had severe wettings lately on repeated occasions and had bad food. Acute pain in several joints. Tonsillitis. Herpes labialis. Temp. 102·3°. Profuse sweats. Sleepless. Sharp pain in precordia; heart sounds normal. Rheumatic pains and tonsillitis abated and disappeared under treatment. On 25th Jan. heart noticed to intermit at every 5th beat. 26th: Slight systolic *bruit* at apex noted. 29th: Systolic *bruit* distinct. 3rd Feb.: Slight relapse of rheumatism, yielding to treatment. 9th Feb: *Bruit* less distinct; it gradually lessened and vanished about the middle of the month. 23rd: Discharged free from rheumatism and with an apparently sound heart.

Case 4. Henry R. 19, coalheaver, Ad. 22 Jan. 1886, Dis. 19 Feb. Case reported by H. P. and clerks.

No previous attack. During 2nd week in Jan. was three times wet through with snow when unable to change. On admission, acute pain in several joints. Temp. 103°. Profuse sweating. First sound at cardiac apex muffled and prolonged. 25th: Pains relieved. Systolic *bruit* at apex. 28th: Pains recurred in some joints. 1 Feb: Pains relieved. Systolic *bruit* less loud. 16th: Cardiac *bruit* has disappeared, the sounds at all areas are normal. Went out on 19th, much too early. Cautioned.

Case 5. Samuel T. 21, Royal Naval Reserve, Ad. 23 Feb. 1886, Dis. 14 April. Reported by H. P. and clerks.

One previous attack of rheumatism. Sustained a severe chill in mid-Atlantic about a fortnight ago. Has pain in most joints. Temp. 102·3°. First sound at cardiac apex is prolonged. 25th: Pains greatly relieved. Continued improving until 9th March when pain recurred and a *bruit* took the place of the 1st sound at the apex. Gradually improved. Murmur diminished and disappeared. Went out quite well and with apparently sound heart on 14th April.

Case 6. Mary A. 16, factory girl, Ad. 3 March, 1886, Dis. 29 March. Reported by H. P. and clerks.

Previous health excellent. Had rigor on 27th February. Vomiting. Acute pain in several joints. Temp. 101·5°. Legs, ankles and face swollen. Dyspnœa and severe cough. Albumen in the urine. Systolic murmur at the apex. Profuse sweating. 5th: Pain much relieved, œdema continues, also sweating. Urine scanty. *Bruit* still heard and loud accentuation of second pulmonary sound. 12th: Murmur becoming feeble. Pains gone. The murmur became less and less day by day and disappeared before the end of the month.

Case 7. Margaret W. 49, housewife, Ad. 13 March, 1886, Dis. 30 April. Reported by H. P. and clerks.

No prior history of rheumatism. Exposed to cold and wet in February, after that had pain in the knees and acute stabbing pain in left side. Much perspiration. Urine scanty. On admission, knees, ankles and wrists swollen and painful. Temp. 101·8°. A systolic *bruit* at the apex. 18th: Much better. *Bruit* less distinct. 19th: Relapse. Temp. 103·7°. *Bruit* very distinct: had two faints. 30th: Heart's action intermittent. Pulse 130. 5th April: Some broncho-pneumonia at bases; cough and dyspnœa. Gradually improved. *Bruit* disappeared and patient went out with apparently sound heart.

Case 8. Ellen L. 19, domestic servant, Ad. 27 March, 1886, Dis. 10 May. Reported by H. P. and clerks.

No previous attack of rheumatism. Illness began on 21st. Pain in joints and some dyspnœa. Temp. 101°. Profuse sweating. Systolic *bruit* at apex. 30th: Much better; *bruit* distinct. 12th: Going on well, but had a slight faint. Improved and gradually lost all trace of *bruit*. Went out with to all appearance a sound heart.

Case 9. John C. 34, labourer, Ad. 23 April, Dis. 24 May, 1888. Reported by Registrar and Mr S. W. Cheetham.

One previous attack of rheumatism. Was much exposed to rain and cold winds a fortnight ago. On admission had severe joint pain. Temp. 101°. Heart normal. Slight albuminuria. Sleeps badly, headache. 26th: Pain subsiding. 30th: A systolic *bruit* has appeared at the base arising apparently from the aortic valves. 18th May: The *bruit* has diminished and disappeared. No albumen in the urine. Sent to Woolton Convalescent Inst. for a fortnight's rest on the 24th May, the heart sounds being normal. At the end of June his heart remained in a normal condition.

Case 10. Anne C. 19, domestic servant, Ad. 10 May, 1888, Dis. 14 June. Reported by Registrar and Mr S. L. Melville.

Has been much exposed to damp. Six weeks ago was first attacked by rheumatism. On admission, pain in wrists, knees and ankles. Temp. 101·2°. Much acid perspiration. Apex beat 4 in. from middle line in 5th space. Dulness slightly increased. Loud and long pre-systolic and systolic *bruits* at apex with much accentuation of 2nd pulmonary sound. Some albumen. 14 May: Pain relieved; cardiac sounds unchanged. 16th: A systolic thrill at apex lasting a few days, then disappearing. 17th: Pre-systolic murmur not heard. 19th: Systolic murmur becoming feeble. 26th: Return of pain in left knee and shoulder. Systolic *bruit* distinct. 1st June: Systolic murmur becoming indistinct. 13th: Murmur now

entirely absent and heart sounds entirely normal. 14th: Sent to Convalescent Institution for a further period of rest.

Case 11. Louisa P. 17, housemaid, Ad. 10 Jan., 1889, Dis. 6 Feb. Reported by Registrar and Mr Stanley Melville.

No prior attack of rheumatism. Had a chill towards the end of December, followed by severe pain in joints. Had previously had dull aching pain about the heart. Apex in sixth space, dulness slightly increased. *Bruit* with first sound at apex heard in axilla. Temp. 100·2°. 18th Jan: All pain gone, *bruit* unchanged. Left hospital on 6th Feb. much too early; *bruit* as on admission.

Case 12. Alice M. 21, general servant, Ad. 9 May, 1889, Dis. 22 May. Reported by Registrar and Mr G. R. Jones.

Strong family predisposition to rheumatism. Patient had acute rheumatism two years ago. Has done much laundry work. Attacked lately with pain in several joints. Temp. 101°. Systolic *bruit* at aortic and pulmonary cartilages, also at apex. Cardiac dulness not increased. Apex in fifth spaces 2½ in. from middle line. 21st: Pains all gone. *Bruits* unchanged. 23rd: Left hospital; much too soon.

Case 13. Mary I. 66, midwife, Ad. 7 June, 1889, Dis. 12 July. Reported by Registrar and Mr G. Aldershaw.

Has had rheumatism before. Present attack began three weeks ago. Pain and swelling in shoulder, knees and feet. Systolic *bruit* at apex. 19th: Pains have disappeared excepting in right foot. The *bruit* disappeared and had been absent for some days when she went out on 12 July. Care and further rest were recommended.

Case 14. Mary McC., servant, Ad. 4 June, 1890, Dis. 16 July. Reported by Registrar and Mr H. W. S. Williams.

No previous attack of rheumatism. Three weeks ago slept in a damp bed. Tonsillitis followed and rheumatic pains. Profuse perspiration. Temp. 102°. First cardiac sound soft

at apex. 2nd pulmonary sound accentuated. 10th June: Much better. 11th: First sound at apex more distinct. 15th: Pains returning in joints. Temp. 102·6°. Tongue foul. Distinct systolic *bruit* at apex. 28th: Much better. 1 July: Temp. 97°. 3rd July: *Bruit* still distinct. 8th: *Bruit* becoming faint. 15th: *Bruit* entirely absent. The need for further care and rest was pointed out.

Case 15. Clara B. 19, dressmaker, Ad. 17 Dec. 1890, Dis. 23 March. Reported by Registrar and Mr W. Ramsay.

Family history of rheumatism. On admission had swelling and pain in several joints. Temp. 101° and 103° at various times. Systolic *bruit* at apex. 2nd pulmonary sound accentuated. Pain disappeared under treatment and in February the *bruit* disappeared also. She had other ailments and was kept in the ward until 23 March and then sent for further rest to the Convalescent Institution. When seen three years later she appeared to be free from all heart symptoms.

Case 16. Anne J. D. 29, housekeeper, Ad. 7 Feb. 1891, Dis. 14 March. Reported by Registrar and Mr C. A. Foyl.

No previous rheumatic history. After a severe cold, pain in the limbs began a fortnight ago. Temp. 102° and 104·4°. Much acid perspiration. Heart sounds normal. Feb. 10th: Delirium. *Bruit* developed at the apex: albuminuria. On 14th pain returned with much severity: delirium. Had vapour bath. 2 March: Much better; *bruit* at apex has diminished and to-day disappeared. Left hospital apparently well on 14 March, being urged to rest for some weeks at home.

Case 17. Mary G. 14, general servant, Ad. 13 May, 1891, Dis. 11 June. Reported by Registrar and Mr S. H. House.

Has had "growing pains" and has been much exposed to damp. A week ago knees and ankles swollen and painful. A systolic *bruit* at apex, 2nd pulmonary sound accentuated. 19th: Pain gone. 24th: The *bruit* has become soft and variable. 2nd June: *Bruit* absent. Went out apparently well on 11th June, further rest being enjoined.

Case 18. Arthur G. 18, porter, Ad. 13 June, 1891, Dis. 13 July. Reported by Registrar and Mr J. B. Maudesley.

No history of rheumatism until last March. Pain became severe in June. Temp. 103°. Cardiac dulness normal. 1st sound at apex muffled. No *bruits*. 17th: A systolic *bruit* has developed at the apex. 25th: Pain nearly gone. 26th: The *bruit* is becoming less distinct. 11th July: *Bruit* entirely absent. 13th: Sent for further rest to Woolton Convalescent Institution.

Case 19. Robert S. 13, school-boy, Ad. 27 Nov. 1891, Dis. 31 Dec. Reported by Registrar and Mr G. Kemball Jones.

No prior attack of rheumatism. Three weeks ago sat in his wet clothes in school and had subsequently a severe attack of rheumatism, which is now (on admission) subsiding. Temp. 100·2°. Distinct systolic *bruit* at apex. Dulness normal. 5th Dec: Pain all gone. 14th: Systolic *bruit* less distinct. 21st: Patient feeling perfectly well. Went out against advice, a systolic *bruit* greatly diminished in loudness was still distinguishable. It was felt that if he could have been induced to remain under treatment and with further rest it might have entirely disappeared.

Case 20. John H. T. 18, window cleaner, Ad. 20 Dec. 1891, Dis. 31 Dec. Reported by Registrar and Mr J. C. Bawden.

Had two prior attacks of rheumatism. After great exposure to wet was attacked on 7th Dec. Temp. 99·5°. Considerable pain in joints. Cardiac dulness normal. Long systolic *bruit* at mitral area: accentuation of 2nd pulmonary sound. 24th: Pain relieved, systolic *bruit* not so distinct. 30th: Feels perfectly well, *bruit* quite absent. 31st: Went home, ought to have remained much longer under treatment. He apparently continued well, however, for after a second attack of rheumatism seventeen months later, when examined nothing but a slight impurity of 1st sound at apex was discovered and he was able to work as a day labourer without any dyspnœa.

Case 21. Thomas P. 28, coachman, Ad. 16 Oct. 1892, Dis. 28 Nov. Reported by Registrar and Mr W. A. Kidd.

No previous attack of rheumatism. After being much exposed to cold and wet was attacked on 7th Oct. with acute rheumatic pains; temp. 100·5°. Cardiac dulness increased; Pericardial friction; a *bruit* at the apex of great loudness. Considerable bronchitis. 25th: Friction disappeared and pain gone. *Bruit* heard intermittently. 23rd Nov.: *Bruit* entirely absent. 28th: Sent to Woolton Convalescent Institution for further rest. Examined him at the end of Nov. 1893: heart perfectly normal. No dyspnœa.

Case 22. Thomas T. 26, canvasser, Ad. Nov. 18, 1892, Dis. Dec. 7. Recorded by Registrar and Mr H. E. Annett.

Attacked a week ago by severe rheumatic pains. Temp. 103·4°. Profuse acid perspiration: cardiac sounds normal, dulness normal. 23rd: Much better, but a distinct systolic *bruit* has appeared at the apex. 26th: *Bruit* less marked. It subsequently disappeared, and he went out on 7th December apparently quite well. The necessity of further rest at home was pointed out to him. He was told to return if any relapse occurred.

Case 23. Andrew F. 28, barman, Ad. 18 Jan. 1893, Dis. 22 Feb. Reported by Registrar and Mr S. J. Ross.

Has had previous attacks of rheumatism: the present began three weeks ago. Much swelling and pain in joints. Temp. 102°. Profuse perspiration. Trace of albumen. Heart sounds normal. 22nd Jan.: Pains subsiding. First cardiac sound at apex becoming prolonged and indistinct. Soon a distinct murmur appeared. Reduplication of 2nd sound; the *bruit* became less marked and on 22nd February had entirely disappeared. Went home and rested until the beginning of April, continuing well excepting slight pain in feet, which began on March 14th. Returned to work on 6th of April, but pains recurred.

Case 24. Andrew F. readmitted on 10th May.

On 1st May pains became very severe and he gave up working. Much pain and swelling in joints. No albumen but some uric acid. Cardiac dulness normal; a distinct systolic *bruit* present again, heard in the axilla. 2nd pulmonary sound accentuated. Notwithstanding treatment the cardiac condition remained thus far unchanged. He went out at his own desire on 30 May free from rheumatism, but not having given himself sufficient rest to secure recovery from the endocarditis.

Case 25. Margaret R. 21, servant, Ad. 25 Jan. 1893, Dis. 20th Feb. Reported by Registrar and Mr T. J. de C. Veale.

Rheumatism commenced two months ago. She had never had it before. Acute pain in back and several joints. Temp. 101°. Cardiac dulness slightly enlarged. No *bruit*, but 2nd sound at pulmonary cartilage accentuated. 26th: A systolic *bruit* has appeared at the apex. 3rd Feb.: Much better, *bruit* distinct. 10th Feb.: First sound at apex nearly normal. 14th: Apex *bruit* absent, but a slight *bruit*, probably hæmic, is heard at the pulmonary cartilage. 20th: Went out apparently quite well. Was sent to the Convalescent Institution for further rest.

Case 26. Ernest P. 35, butler, Ad. 11 March, 1893, Dis. 20 April. Reported by Registrar and Mr T. J. de C. Veale.

Attacked by rheumatism a month ago for the first time, improved, but got a chill on 4th March and was worse again. Much pain in joints. Temp. 102°. Heart sounds normal, but apex 4 inches from middle line. 14th March: Systolic *bruit* developed at apex, also signs of pericarditis. Dulness extends up to 2nd space. 17th: Dulness diminishing. Pain almost gone. 24th: Relapse of rheumatism, further effusion into pericardium. Sounds at apex indistinct. Accentuation of 2nd sound at base. 26th March: Signs of pneumonia and pleuritic effusion at left base. 29th: Improving, cardiac dulness less. First sound clear, no *bruit* at apex. A diastolic *bruit*, the

nature of which was somewhat obscure, developed at the pulmonary valve, where the 2nd sound continued to be accentuated. 6th April: Cough and lung dulness gone. No apex *bruit*. 20th April: Went out quite well, but was advised to rest for a time at home.

Case 27. Esther R. 30, housewife, Ad. 17 May, 1893, Dis. 23 June. Reported by Registrar and Mr J. A. Swainson.

Had one prior attack of rheumatism. On admission had pain in all her joints. Temp. 102°. Discomfort in cardiac region. Heart apparently normal. Profuse sweats. 23rd: A distinct systolic *bruit* at the apex to-day. 6th June: Much better. *Bruit* unchanged and 2nd pulmonary sound is accentuated. *Bruit* became softer, and on 19th June had entirely gone. 23rd June: Left hospital apparently quite well. The need for further rest at home was pointed out to her.

Case 28. Daniel H. 19, labourer, Ad. 19 July, 1893, Dis. 17 Sept. Reported by Registrar and Mr W. E. Tarbet.

Two previous rheumatic attacks. Has much pain in several joints. Temp. 101·7°. Heart normal. 21st July: Pain yielding. 22nd: A distinct systolic *bruit* heard at apex. 9th Aug.: *Bruit* less distinct. 21st Aug.: Relapse of rheumatism; temp. 100·7°. The *bruit* has become more distinct: 2nd pulmonary sound accentuated. Treatment was continued, but no change in the *bruit* took place. He left hospital on 17th Sept. Nothing further has been heard of him. It appeared probable that the treatment had failed.

Case 29. Mary McK. 16, servant, Ad. 9 June, 1893, Dis. 7 July. Reported by the Registrar and Mr J. A. Swainson.

Patient has suffered from sore throat and bronchitis frequently. No distinct history of rheumatic pains until three weeks ago. Pain severe on admission. Temp. 101·8°. Much perspiration. Loud systolic *bruit* at apex, heard in axilla 2nd pulmonary sound accentuated. She has long suffered from

palpitation. 12th June: Much better. Gradually improved, but no change occurred in the heart sounds. It appeared probable that the cardiac mischief was of long standing.

Case 30. Agnes D. 42, cook, Ad. 11 Jan. 1894, Dis. 3 March. Reported by Registrar and Mr F. S. Rowland.

Strongly rheumatic history. Several painful and swollen joints on admission. Temperature normal. Tonsillitis. Systolic *bruit* at the apex. No accentuation of 2nd pulmonary sound is recorded. She gradually improved; the *bruit* disappeared. She went out in good health on 3rd March. Heart apparently quite sound. Prolonged rest enjoined.

Case 31. Elizabeth P. 19, servant, Ad. 2 May, 1894, Dis. 30 May. Reported by the Registrar and Mr J. Johnson.

No rheumatism prior to end of March. Severe joint pains on admission. Temp. 100·4°. Cardiac dulness normal. A systolic *bruit* at the apex. Improved under treatment, lost all pain. The *bruit* entirely disappeared. She went home for further rest.

Case 32. Charles C. 21, butcher. Ad. 24 June, Dis. 18 Aug. 1894. Reported by Registrar and Mr J. Johnson.

No previous illness of any importance. Present attack began a week ago after sleeping in a damp bed. Pains in large joints; bad cough: temp. 102·4°. Cardiac dulness increased. Pulse 98: a rasping systolic *bruit* heard at the apex. Respiration 36. Pneumonia on the right side. Trace of albumen. 26th: Rheumatic pains gone. 28th: Pericarditis present, friction and effusion. 2nd July: Temp. 101°. Pulse 112. Respiration 48. Pain referred to cardiac region. Rusty sputum. Slight effusion into right pleura. 5th July: Cardiac dulness diminished. Double friction loud. Resp. 32. Blisters and poultice had been applied over precordia. 9th July: Pneumonia subsiding. 11th: Pulse 88. Resp. 28. No albumen. Improved steadily from this time; friction rub and also the apex *bruit* became indistinct. The *bruit* disappeared entirely early

in August. On 18th August was discharged. No trace of *bruit* remaining. Was sent to the Convalescent Inst. for further rest and cautioned to avoid exertion for some months to come.

Case 33. Emily J. 17, servant, Ad. 4 July, 1894, Dis. 13 Aug. Reported by Registrar and Mr J. Johnson.

No previous rheumatism. Got wet a week ago and was attacked by pain in wrists, ankles and knees. Temp. 102·3°. Heart sounds normal. Has some bronchitis. 13th July: Pain gone, but an accentuation of pulmonary 2nd sound is observed. 16th: A systolic *bruit* heard at the apex. *Bruit* gradually became soft, then variable and disappeared. Went out apparently well on 13 Aug. Sent to Convalescent Institution for further rest.

Case 34. Rachel E. 15, servant, Ad. 4 July, 1894, Dis. 28 July. Reported by Registrar and Mr E. P. Mead.

No previous rheumatism. Has vague pain in the cardiac region and in abdomen, swelling in joints of legs and feet and tonsillitis. Temp. 104·3°. Cardiac dulness increased slightly upwards and to the right. A systolic *bruit* at the apex also heard at pulmonary cartilage. Slight dulness and crepitation at the bases of the lungs. Pericardial friction heard over right side of the heart. Under treatment the cardiac and other symptoms passed away, viz., dulness decreased, friction and apex *bruit* disappeared, and she went home on 28th July with apparently a sound heart, further rest and quiet being enjoined.

Case 35. Madeline F. 18, barmaid, Ad. 14 June, 1894, Dis. 9 Aug. Reported by Registrar and Mr J. Johnson.

No distinct history of rheumatism, though she has had tonsillitis and probably "growing pains." Present rheumatic attack began three weeks ago. Joints are swollen and painful. Temperature normal. A systolic *bruit* at the apex heard in the axilla and reduplication and accentuation of 2nd pulmonary sound. Cardiac dulness normal. Impossible to decide whether

cardiac lesion is recent or of long standing. Some albumen. Knee jerks increased; a papillary rash on the trunk. 18th June: albumen gone. Pain better in the legs. Elsewhere rheumatic pains lasted for a month in spite of treatment. Gradually improved, but did not lose the apex *bruit*. Went out 9th August. Heart in *statu quo*.

Case 36. Ellen K. 24, ward maid, Ad. 25 July, 1894, Dis. 3 Oct. Reported by the Registrar.

No previous rheumatism. Illness began three weeks ago. Several joints swollen and painful on admission. Heart's action irregular. No marked increase of cardiac dulness. Impulse in 5th space just within nipple line. Systolic and pre-systolic *bruits* at apex; accentuation of 2nd pulmonary sound. Systolic *bruit* at the aortic. Has suffered from dyspnœa on exertion for a considerable time. 27th July: Pains relieved; heart sounds the same. 14th Aug.: Recurrence of rheumatism. Temp. 101°. Pain and fever continued for some days, then abated. 11th Sept.: A second recurrence of pain, temp. 101·4°. A loud diastolic *bruit* developed some days ago at the aortic cartilage. 25th Sept.: Now feels well. Presystolic thrill, also presystolic and systolic *bruits* at apex and accentuation of 2nd pulmonary. Aortic *bruit* absent. Went out 3rd Oct. The mitral disease was probably of long standing. The aortic mischief seems to have been recent. It subsided under treatment.

Case 37. Mary A. H. 22, shop girl, Ad. 8 Dec. 1894, Dis. 16 Jan. Reported by Registrar and Mr J. Atkinson.

Rheumatic family history, but patient was never attacked until a fortnight ago. On admission, several joints swollen and painful. Temp 99·2°. Cardiac dulness normal. A systolic *bruit* at the apex, 2nd pulmonary sound accentuated. 17th Dec.: Pains gone, *bruit* distinct. Towards the end of the month the *bruit* became variable and was absent on 12th Jan. Went out on 16th apparently quite well—to have further rest at home.

Case 38. James B. 38, barman, Ad. 18 March, 1894, Dis. 10 April. Reported by Registrar and Mr F. S. Rowland.

No previous rheumatism. Has of late years taken "nips," though not habitually intemperate. Has had rheumatism for a fortnight. Temp. 100·4°. Slight icterus. Pulse rather tense and irregular, bigeminal in character. Apex beat in 5th space four inches from middle line. Dulness normal, heart beats in couples. A strong distinct systolic *bruit* heard with the first of the twin systoles, heard in axilla. Slight albuminuria. The pains were much better on 21st March. Pulse is becoming regular and losing its bigeminal character. 3rd April: The *bruit* has disappeared. 10th April: Went out quite well. Cautioned to avoid exertion, alcohol, and exposure to cold.

Case 39. John G. 27, dock labourer, Ad. 17 Oct. 1894, Dis. 6 Dec. Reported by Registrar and Mr H. M. Crake.

No distinct history of previous rheumatism; takes much alcohol. After exposure to cold and wet a week ago had pain and swelling in several joints. Temp. 101°. Was bathed in acid perspiration when brought to the hospital. Cardiac apex displaced outwards. A booming systolic *bruit* replaces first sound at apex, second reduplicated at pulmonary cartilage. 25th Oct.: Pains relieved. Gradually lost all rheumatic pains and left hospital on 6th Dec., the cardiac *bruit* being unchanged.

Case 40. Rose P. 12, school girl, Ad. 6 Feb., 1895, Dis. 9 March. Reported by Registrar.

Had one previous rheumatic illness. Now suffering from chorea. Heart normal. 15th Feb.: First cardiac sound at apex becoming soft and indistinct. 16th: Temp. 103·2°. 17th: Temp. 103°. 19th: A distinct systolic *bruit* at apex. Temp. 101·4°. 21st: Better, temp. 97°. 1st March: All choreic movements gone, *bruit* distinct; but after this date it subsided and disappeared. She went home on 9th March with apparently a sound heart. Examined her heart in Feb. 1896 and found it perfectly normal.

Case 41. Noah G. 43, coal trimmer, Ad. 16 Jan. 1895, Dis. 18 Feb. Reported by Registrar and Mr W. A. Kidd.

Patient never had rheumatism before, though a strong family tendency to it exists. Attacked by joint pains three weeks ago, had them still on admission. Temp. 101·5°. Profuse acid perspiration. Apex beat in 5th space in nipple line. Systolic *bruit* at apex, conducted into axilla. 22nd: Pain still severe. 26th: Temp. 103·2°. Salicylates appear to cause vomiting; their administration stopped; the joints were blistered. 28th: Pain much relieved. Temp. 100·5°. Blood examined, much leucocytosis. 7th Feb.: Pain in joints gone; Temp. normal. Cardiac *bruit* has been diminishing in loudness 12th: No *bruit* to be heard. Left hospital on 18th with apparently a sound heart. Instructed to avoid exertion for some weeks to come.

Case 42. Alice P. 18, servant, Ad. 4 March, 1895, Dis. 15 May. Reported by Registrar and Mr F. H. Evans.

Had previous attack of rheumatism. Present attack began a month ago. Has pain in several joints. Temp. 102·8°. Cardiac dulness slightly increased upwards and to the right. Systolic *bruit* at apex. Accentuation of 2nd pulmonary sound. 8th March: Pain relieved. The dulness (due to pericardial effusion?) is now lessened. Gradually improved. *Bruit* became soft and variable on 22nd April, absent on 29th. First sound soft. The accentuation of the 2nd pulmonary continued for some time. 15th May: Went out with apparently sound heart—to have further rest at home.

Case 43. Alice P. 18, readmitted on 1 Oct., Dis. Nov. 19, 1895.

She continued perfectly well from 15th May until the end of August, when she was again attacked by rheumatism. Several joints now affected. Temp. 102°. During the past month she had had no treatment other than rubbing the joints with a liniment. Cardiac dulness normal. A systolic *bruit* at the apex. Conducted into the axilla. Pulmonary 2nd

sound accentuated and reduplicated. 7th Oct.: Pains gone and temperature normal. *Bruit* unchanged. 16th: Feels well. Treatment continued until 12th Nov. when patient went out otherwise quite well, but with evidence of mitral disease.

Case 44. John G. 14, messenger, Ad. 17 July, 1895, Dis. 16 Sept. Reported by Registrar and House Physician.

No prior rheumatism. Present pains in joints began only two days ago. Temp. 99°. Cardiac dulness increased towards middle line and upwards. Precordia bulges slightly. Systolic *bruit* at apex heard in axilla. Accentuation and reduplication of 2nd pulmonary sound. A peculiar roaring sound audible at pulmonary valve. Under treatment the rheumatism soon disappeared, but although the treatment was continued until the middle of September the apex *bruit*, though greatly lessened in volume never disappeared and the pulmonary accentuation still existed. Disease thought to be probably of long standing, possibly congenital.

Case 45. John C. 23, carter, Ad. 12 Oct., 1895, Dis. 19 Dec. Reported by Registrar and Mr A. F. Seacombe.

Had rheumatism two years ago. Attacked by acute pain in several joints a week ago. Temp. 104·4°. Profuse acid perspiration. Sordes on lips, blanket-like fur on tongue. Diarrhœa. Pulse 126, weak. Cardiac dulness increased. Pericardial friction. Respirations between 40 and 60, *râles* and crepitation so loud that it is difficult to say whether the heart sounds are pure or not. Patient continued extremely ill for several weeks. Had an enteric rash, stools of a pea soup type, a temperature and sphygmogram of enteric character until the 4th Nov. The rheumatic pains and swelling about the joints subsided about the 21st Oct. Had double pneumonia, much delirium, and on several occasions Cheyne-Stokes breathing. Patient became so extremely weak that his life was despaired of, but he always took fluid nourishment and stimulants well. On 11th Nov. patient improving, a systolic *bruit* was heard at the apex. Respiratory sounds are now less

loud. Accentuation of 2nd pulmonary sound. Friction still audible. 18th: Cardiac dulness diminishing. From this time all other symptoms gradually subsided excepting those indicating mitral disease. Sent to Woolton Convalescent Inst. on 19th Dec. Examined in June 1898. Has had another attack of acute rheumatism. Has marked mitral regurgitation.

Case 46. Richard C. 15, errand boy, Ad. 20 Nov. 1895, Dis. 25 Jan. 1896. Reported by Registrar and Mr H. M. Henderson.

Had rheumatism before. Present severe pains in joints of arms and legs began a week ago. Four days ago choreic movements commenced. Cardiac dulness normal. No *bruits*, but first sound at apex is soft and weak. 26th Nov.: Otherwise improving, but a systolic *bruit* has appeared at the apex and the 2nd pulmonary sound is accentuated. 27th: *Bruit* heard in the axilla. 2nd Dec.: Chorea almost gone. 18th Dec.: Patient almost well, but *bruit* is heard. 28th: The *bruit* has changed, becoming progressively softer and less distinct. 2nd Jan.: *Bruit* scarcely audible, after that disappeared. On 25th Jan. patient went out with apparently a sound heart. Was cautioned to avoid exertion for some time to come.

Case 47. Henry G. A. 29, window cleaner, Ad. 23 Nov. 1895, Dis. 3 Jan. 1896. Reported by Registrar and Mr E. E. Laslett.

Has had three severe attacks of acute rheumatism and other slighter attacks. About middle of November slept in a damp bed. Severe joint pains commenced on 17th Nov., also bronchitis. Temp. 103°. Cardiac dulness normal. Systolic *bruit* at apex. Pulmonary 2nd sound accentuated, has been breathless for the last few days. Has bronchitis, pneumonia, and slight pleurisy on right side. 20th: Pains relieved, friction gone. Temp. 100·2°. Albuminuria. 10th Dec.: Much better. The *bruit* has disappeared but the first sound at the apex is soft and weak. 20th: A slight return of rheumatism, and of the apex *bruit*. 2nd pulmonary accentuation distinct. Temp. 101°.

23rd: Pains relieved and *bruit* almost absent. Pulmonary accentuation very slight. 27th: *Bruit* entirely absent. 3rd January went out, heart apparently normal. Cautioned as to rest and avoidance of causes of rheumatism.

Case 48. Henry G. A. 29, readmitted 19 Feb. 1896, Dis. 16 April. Reported by Registrar and Mr J. E. Smith.

It appears that patient had only been home about ten days, when the pains recurred. He was treated at home and improved, but got worse about the 1st of Feb. When admitted on 19th, had pain and swelling in several joints. Cardiac dulness normal. Systolic *bruit* at apex. Has some bronchitis. Patient improved steadily. On 2nd March the *bruit* had become very faint. On 11th it became variable, and after the 27th was never heard at all. On 16th April was sent to Woolton Convalescent Inst. for further rest, the heart being apparently sound.

Case 49. Harriett H. 16, laundress, Ad. 5 Feb. 1896, Dis. 9 April. Reported by Registrar and clerks.

No previous rheumatism, though patient had been much exposed to damp and cold. Pain and swelling in several joints. Temp. 101·2°. Cardiac dulness a little increased. A systolic *bruit* at apex, heard in axilla. Accentuation of 2nd pulmonary sound. 10th: Pain much better. Considerable amount of oxalic acid in urine. Patient improved in every respect excepting as regards heart sounds. Was sent to Woolton on 9th April quite well but having a systolic *bruit* as on admission.

Case 50. Frances M. 9, school girl, Ad. 29 June 1896, Dis. 18 Aug. Reported by Registrar and Mr J. Bradley Hughes.

Has had growing pains and sore throats. States that she has habitually got breathless when running or playing, more so than other girls did. Now has acute rheumatism, several joints swollen and tender. Temp. 101·6°. Cardiac dulness rather increased in vertical direction, not transversely, impulse feeble. Systolic *bruit* at apex, audible in axilla. Accentuation and

occasional reduplication of pulmonary second sound. Pains and fever subsided in three days, but the cardiac signs remained, the *bruit* apparently less loud than on admission. She was discharged on August 18th. This case was probably one of some standing, but we resolved to give patient the chance of any benefit the treatment might offer.

Case 51. Mary D. 14, school girl, Ad. 11 Dec. 1895, Dis. 13 January. Reported by Registrar and Mr D. G. Hurter.

Had a previous attack of rheumatism, present attack began five weeks ago. Slight occasional systolic thrill at apex. Apex beat in 5th space 3 in. from mid-line. Systolic *bruit* at apex, reduplication of 2nd sound at pulmonary. Pulse 96, slightly irregular in time. At present has slight dyspnœa on exertion, pain and fever subsided, and towards the end of the month the *bruit* lessened and was absent on 31st Dec. She was kept in hospital for a fortnight longer, and then discharged apparently quite well. Sent for further rest to Convalescent Inst.

Case 52. James B. 20, barman, Ad. 7 March, 1896, Dis. 23 April. Reported by Registrar and Mr L. S. Whitwam.

Patient is a total abstainer. No previous rheumatism. Present attack began a fortnight ago. Pains in several joints. Temp. 100·4°. Cardiac dulness normal. Systolic *bruit* at apex, conducted into axilla. Under treatment pain disappeared. *Bruit* became soft about 2nd April, then showed variability and finally disappeared. Dismissed on 23rd April for further rest at Woolton Conv. Inst. his heart appearing to be absolutely normal.

Case 53. John T. J. 14, quarry worker, Ad. 28 Feb. 1896, Dis. 11 April. Reported by Registrar and Mr S. C. Moore.

Has had chorea before. No distinct history of rheumatism. Present choreic attack began after an accident. No history of dyspnœa. Cardiac dulness normal. Systolic *bruit* at apex,

conducted into axilla. Accentuated 2nd sound at pulmonary, pulse being irregular in time. Chorea treated by arsenic. On March 17th, *bruit* diminishing markedly in loudness, then became variable, and entirely disappeared before 24th. Went out on April 5th with apparently sound heart. Ordered to avoid all exertion. In this as in several other cases efforts to trace the patient and learn his subsequent history failed.

Case 54. Peter R. 38, dockgate man, Ad. 10 March, 1896, Dis. 23 April. Reported by Registrar and Mr F. Lovegrove.

One previous attack of rheumatism. No dyspnœa. Frequently gets wet through. Present illness began three weeks ago. Pain in most joints. Temp. 101·8°. Profuse acid sweats. Cardiac dulness normal, 1st sound at apex slightly prolonged, no *bruit*, accentuation of 2nd pulmonary sound. Trace of jaundice. Small amount of albumen. 20th, Pain much relieved. 23rd, a distinct *bruit* systolic in time at the apex to-day. The *bruit* became less distinct, and had disappeared by the 8th April. Was sent to Woolton Convalescent Inst. on 23rd April for a further period of rest. Heart perfectly normal.

Case 55. Mary L. 17, nursemaid, Ad. 21 June, 1896, Dis. 25 July. Reported by Registrar and Mr K. F. Lund.

No previous history of rheumatism. Swelling and tenderness in ankles and knees. Temp. 99·8°, later 102°. No increase of cardiac dulness. Systolic *bruit* at apex. Accentuation of 2nd pulmonary sound, pulse 108. Trace of albumen, abundance of uric acid crystals. *Bruit* became indistinct during 1st and 2nd week of July, and finally disappeared. Was sent home for further period of rest.

Case 56. Annie H. 19, factory-girl from Leicester, Ad. 5 Aug. 1896, Dis. 21 Nov. Reported by Registrar and Mr T. T. Bark.

Had suffered from rheumatism once before. Has had dyspnœa. Much pain and swelling in joints. Temp. 103°.

Pulse 108. Profuse acid perspiration. First sound at apex soft and murmurish. 8th: Pains and swelling gone. A soft systolic *bruit* at apex, heard also in axilla. Temp. normal. 13th: An occasional presystolic murmur at apex in addition to the systolic. 17th: Systolic *bruit* loud and distinct, presystolic absent. 3rd Sept.: *Bruit* much less distinct, but accentuation of 2nd pulmonary sound exists. 21st Oct.: The *bruit* can still be heard though not over so wide an area as before. Towards the middle of Nov. the *bruit* finally disappeared, and the patient went home to have a period of prolonged rest.

Case 57. Margaret S. 18, shopgirl, Ad. 28 Nov. 1896, Dis. 30 Dec. Reported by Registrar and Mr I. T. Grierson.

No rheumatism until ten weeks ago, when patient got wet through and remained all day in wet clothing. Had been under homeopathic treatment ever since without any benefit. Has pain in several joints. Cardiac dulness normal. Systolic *bruit* at apex heard in axilla, reduplication of second sound at base; first aortic sound rough. Trace of albumen. 2nd Dec.: Pain gone, now for first time can move her limbs with freedom. 10th Dec.: Accentuation of 2nd pulmonary sound. 14th Dec.: *Bruit* became variable some days ago and has now disappeared. 30th Dec.: Went out with apparently a sound heart. Was instructed to rest for a considerable time at home.

Case 58. Florence M^{c}D. 18, servant, Ad. 20 Feb. Dis. 20 May, 1897. Reported by Registrar and Mr S. M. Green.

No history of previous rheumatism. Attack began a week ago. Several joints affected. Temp. 100°. Pulse 76, irregular in time. Cardiac dulness normal. A systolic *bruit* at apex, heard in axilla, loud and distinct. 23rd: much better, but *bruit* distinct. During the early part of March the *bruit* was observed to become progressing softer, and on the 19th it was found to have entirely disappeared. Still a tendency to slight rheumatic pains, but heart sounds remained normal. On April 26th had severe relapse of rheumatism. Temp. 102·6°: which

rapidly gave way before large doses of salicylates. The first sound at the apex became less clear, but no distinct *bruit*. A hæmic *bruit* developed at the pulmonary cartilage. Was sent to the Convalescent Institution for further rest on 20th May, the apex sound being quite normal. Seen on 27 July, 1900. Is anæmic, but heart appears quite normal.

Case 59. Annie T. 19, servant, Ad. 15 March, Dis. 29 April, 1897. Reported by Registrar and Mr T. G. Churton.

Previous health indifferent, much exposed to damp and cold. Has had rheumatism for a month. Knees and other joints swollen and painful. Temp. 101°. Cardiac dulness slightly increased upwards and to the left. Systolic *bruit* heard at all areas and in the axilla. Accentuated 2nd sound at pulmonary. It was feared that the cardiac mischief in this case was of long standing; the treatment was tried until 29th April but without result. Heart remained in *statu quo*.

Case 60. Catherine K. 19, general servant, Ad. 24 Sept. 1897, Dis. 18 Oct. Reported by Registrar.

One previous attack of rheumatism. Has had slight rheumatism for nearly three months, it became severe on 21st. Has pain and swelling in several joints. Temp. 101·1°. Has some palpitation and a little dyspnœa on exertion. She is however rather anæmic. Apex in 5th space $3\frac{1}{4}$ inches from mid line, a somewhat heaving impulse. Dulness not increased. A presystolic thrill at the apex. A presystolic *bruit* at apex and also a blowing systolic, both heard in axilla. A loud rough systolic *bruit* heard both at pulmonary and aortic valves. Accentuated second pulmonary sound. The rheumatic pains soon yielded. Towards the end of the month the thrill became feeble and the apex *bruits* soft and difficult to make out; the base *bruits* being thought to be in part at least anæmic, iron was given. All trace of *bruit* gradually subsided, and the patient became to all appearance perfectly well. She was sent on 18th October to Woolton Convalescent Inst. for more prolonged rest. This case is in the writer's experience quite

unique. It seemed as though some very serious constricting condition of the mitral valve subsided under rest and treatment. Within a comparatively short period of time the cardiac trouble apparently disappeared. The patient unfortunately has disappeared also and it has not been possible to trace her later history.

Case 61. John S. 28, stoker, Ad. 14 Dec. 1897, Dis. 21 Feb. 1898. Reported by Registrar and Mr S. M. Green.

Had rheumatism when 16, and an acute attack three years ago. Present illness began a fortnight ago. Pain and swelling in several joints. Temp. 99·4°. Cardiac dulness normal. A systolic *bruit* audible on the inner side of the apex towards lower end of sternum. The pains did not yield as fully and readily as usual under treatment by salicylates and alkalis. Blisters and lead and opium applied to some joints. Temp. rose on 26th to over 101°. The *bruit* became soft and on 1st Feb. had finally disappeared. Was sent to Convalescent Inst. on 21st Feb. for further rest.

Case 62. John B. 14, school-boy, Ad. 12 Nov. 1897, Dis. 12 Jan. Reported by Registrar and Mr G. P. Barff.

Excellent health hitherto. After a wetting was seized with pains in the joints a fortnight ago. Temp. 102°. Pulse 122. Cardiac dulness normal. Slight epigastric pulsation. Systolic *bruit* at apex. 2nd pulmonary accentuated. Slight chorea. Cloud of albumen. 16th Nov.: Pain and fever gone, less choreic movement. 18th: *Bruit* louder. 6th Dec.: Murmur becoming very soft. 19th Dec.: Very difficult to hear the *bruit*; disappeared entirely and was not heard again. Returned home on 12th Jan. Was advised to avoid exposure and exertion. If he ran up a flight of stairs he had some slight breathlessness, more than he would have had prior to the illness.

Case 63. John B. 14, readmitted on 25th Feb.

He had been exposed to cold, and had some catarrh and traces of chorea. A slight systolic *bruit* was heard at apex.

26th: Pulse 75 regular. Temp. normal. Impulse seen in 4th and 5th spaces in nipple line. Superficial and deep cardiac dulness normal. A soft blowing systolic *bruit* at apex extends throughout the systole. Not audible in axilla. Much accentuation of 2nd pulmonary sound. *Bruit* to be heard in tricuspid area. Uric acid in urine. During March chorea became worse and was treated by arsenic. Temp. rose on 9th April to 101·2°. The chorea had disappeared on 11th May. Cardiac *bruit* at apex still manifest. As patient had been overtime in hospital he was sent to rest at home, the *bruit* still existing on 2nd June; blistering &c. had been continued up to this date. While at home the *bruit* gradually got less and finally disappeared altogether. The importance of continued rest had been strongly urged. He was examined at intervals. Heart remained sound. Last seen in July 1900. He has grown tall, is in excellent health, works hard at a laborious trade and has no dyspnœa. Sounds are normal, but the impulse is slightly increased and there is probably a small degree of hypertrophy.

Case 64. Jane E. D. 14, bookbinder, Ad. 1 Feb. 1898, Dis. 4 July. Reported by Registrar and Mr G. P. Barff.

Has had indifferent health. Works in a cold and draughty shop. Three weeks ago attacked by acute rheumatism. Has suffered lately from palpitation. Has pains in several joints. Temp. 103°. Pulse 96. Cardiac dulness normal. A systolic *bruit* at the apex, heard in axilla. Marked accentuation of 2nd pulmonary sound. A soft systolic *bruit*, probably hæmic, heard at pulmonary. The pain and fever soon disappeared. 17th: Right sided facial paralysis has occurred. Heart sounds unchanged; dilatation of right pupil. 28th: The facial paralysis is disappearing. At beginning of March the *bruit* became soft and the pulmonary accentuation less marked. 5th: Paralysis almost gone. At beginning of April the *bruit* became so feeble as to be heard with great difficulty and finally disappeared. She was still kept at rest in hospital.

On 12th May she was unfortunately again attacked by rheumatism, and on the 14th the *bruit* was again heard. On

27th all pain, swelling and stiffness of joints had disappeared, but the *bruit* remained. The treatment was continued until the end of June, when she was sent to the Convalescent Inst. for further rest, the *bruit*, though become slight, being still present; it disappeared later. Examined her on 8th May, 1900. No trace of *bruit*. Heart seems perfectly normal excepting that some accentuation of 2nd pulmonary sound exists. No palpitation. No dyspnœa on exertion. She works hard and enjoys perfect health.

Case 65. Alfred T. 34, labourer, Ad. 17 June, 1898, Dis. 20 July. Reported by Registrar and Mr C. R. Moss.

Marked family history of rheumatism. No definite history of rheumatism in himself until the spring of the present year. A history of alcoholic excess. Has been suffering from rheumatism for some time prior to admission. Has pain and swelling in several joints. Temp. 102°. Cardiac dulness normal. Systolic *bruit* at apex, audible also at pulmonary cartilage and lower end of sternum. Has considerable bronchitis.

The rheumatism subsided completely under treatment but the patient could not be induced to remain long enough in hospital to give a fair chance to the treatment employed for the heart: the *bruit* was distinct when he was discharged on the 20th July.

Case 66. John T. 24, seaman, Ad. 12 July, 1898, Dis. 26 July. Reported by Registrar and Mr T. R. Gornall.

Six weeks ago left the sea and became a climber and wire fixer for the Telephone Co. Spending most of the day at the top of telephone poles he is much exposed to the weather and has often got wet. A month ago seized with rheumatic pains. Had previously been healthy. On admission severe pains and some effusion in several joints. Temp. 101·6°. Profuse sweats. Cardiac dulness not increased upwards or to the right but extends four inches from mid-line on the left. No thrill. A blowing systolic murmur at apex. Accentuation of 2nd pulmonary. Has some bronchitis. Under treatment the rheumatic symptoms rapidly disappeared. At an early date the

bruit became soft and gradually disappeared. He felt so well that he insisted in going out on 26th July, although prolonged rest was thought highly desirable for him.

Case 67. Frederick G. 26, butcher, Ad. 16 Sept. 1898, Dis. 8 Nov. Reported by Registrar and Mr J. S. Byrne.

Good health previously. After a severe wetting was attacked by acute rheumatism a week ago. Several joints painful and swollen on admission. Temp. 102°. Heart normal, no *bruit* of any kind. Trace of albumen. 18th: Still considerable pain. A systolic *bruit* has developed at the apex. 2nd pulmonary sound accentuated. 19th: Temp. 102·6°. 20th: Temp. 103°. 21st: Temp. 100·6°. 22nd: Temp. 99·2°. Pains almost gone. 27th: Pain absent, *bruit* loud. 6th Oct.: *Bruit* becoming fainter. Albumen gone. On 3rd Oct. the salicylates were stopped, the other treatment being continued. On 7th Oct. temp. 100·5°, and on 9th 101° and return of pain. 15th: Pain gone. *Bruit* audible. 27th Oct.: *Bruit* not always heard. 4th Nov.: *Bruit* scarcely discoverable. Appears quite well. Went out on 8th. Heart apparently sound. Was cautioned to avoid exertion for some time to come.

Case 68. Louisa E. 35, cook, Ad. 24 Jan. 1899, Dis. 24 April. Reported by Registrar and Mr C. W. Budden.

One previous attack of rheumatism. Present attack began a fortnight ago. Much pain in several joints. Temp. 102·4°. Profuse perspiration. Cardiac dulness extends to right border of sternum, other limits normal. Loud harsh systolic *bruit* at apex, heard in axilla and in all areas. 2nd pulmonary sound accentuated. 30th: Much better. Pains nearly gone. 3rd Feb.: Sickness, the *bruit* has become soft; impulse has a peculiar double character. 6th: Pneumonia on left side. Temp. 103°. An occasional systolic thrill felt. 11th: Temp. 103·4°. Some delirium. Respiration 40. Tenderness in some joints. 14th: Albumen in urine. Respiration at one time 60. 16th: Still very ill, respiration 44. Involuntary evacuations. 24th: Improving. Systolic *bruit* still distinct. 4th March: Much improvement. 15th: *Bruit* has become very feeble. 17th:

Bruit quite absent when patient sits up. Scarcely perceptible when recumbent. 25th March: No trace of *bruit*. Sent to Woolton Convalescent Inst. on 24th April. No sign either of *bruit* nor of pulmonary accentuation. She was cautioned to avoid exertion. Her heart was examined also on 29th June and appeared to be perfectly sound.

Case 69. Annie G. 23, housewife, Ad. 24 March, 1899, Dis. 11 May. Reported by Registrar.

A rheumatic family history. Patient has had four previous attacks. Present attack began last Christmas. Pain and swelling in several joints. Temp. 101·5°. Cardiac dulness normal. First sound soft. Second accentuated at the pulmonary valve. The soft first sound changed and a distinct systolic *bruit* appeared. On May 5th the *bruit* had disappeared. Her heart was apparently healthy when she was discharged on 11th May. She was cautioned to avoid exertion for some weeks.

Case 70. Robert G. 8, schoolboy, Ad. 27 April, 1899, Dis. 11 June. Reported by Registrar and Mr P. J. Caddow.

Patient had acute rheumatism three months ago. Present attack less severe. Temp. 101·4°. Cardiac dulness increased to left and slightly to right. Blowing systolic *bruit* at apex conducted to axilla. Marked accentuation of 2nd pulmonary sound. A faint systolic *bruit* heard at aortic area. *Bruit* heard loudly at the back. Difficult to ascertain whether the cardiac mischief was of long standing or not. Probably it was. Treatment was however attempted on the chance of its being recent. All sign of rheumatism disappeared, but the cardiac condition remained unchanged. Discharged on 11th June.

Case 71. John C. 30, dock labourer, Ad. 4 April, 1899, Dis. 29 May. Reported by Registrar and Mr G. W. Gelderd.

Patient had rheumatism last summer. Present attack began nine weeks ago. Several joints swollen and painful. Temp. 103·2°. Pulse 74. Cardiac dulness slightly increased

to the left. No *bruits* but first sound at apex is soft and indistinct. 2nd pulmonary sound accentuated. A *bruit* developed at the apex a few days after admission. After the usual treatment the pain and swelling subsided, and on 1st May the *bruit* had become indistinct. It had entirely disappeared on the 15th. His heart appeared to be perfectly normal when he was sent to the Woolton Convalescent Inst. on the 29th May for a further period of rest.

Case 72. Mrs S., a patient in a private ward in the Infirmary.

Had suffered from acute rheumatism for some days. First cardiac sound soft on admission on 23rd May, 1899. A distinct systolic *bruit* heard on the 27th with accentuation of 2nd pulmonary sound. Under treatment the rheumatism rapidly subsided and within a fortnight the *bruit* became soft and irregular and finally disappeared, together with the accentuation of the 2nd pulmonary sound. She returned to her home in the Midland Counties on the 19th June, her heart being apparently sound, after being cautioned to avoid all exertion for six or eight weeks. 23rd May, 1900: Patient reports herself to be in perfect health having no dyspnœa nor any sign of heart disease. I had no opportunity of examining her personally.

Case 73. Theresa C. 19, laundress, Ad. 16 May, 1899, Dis. 30 July. Reported by Registrar and Mr P. J. Caddow.

Family history of rheumatism, but no clear evidence of a prior attack in case of patient. Present illness began a fortnight ago when she remained in wet clothing for seven hours. Severe pain in several joints; temp. 102·3°. Cardiac dulness considerably increased upwards and to the left and right. A blowing systolic *bruit* at the apex, conducted into the axilla. 2nd pulmonary sound accentuated. 19th: Pains better, temperature fell to normal. Cardiac mischief thought to be probably of long standing, but treatment nevertheless was attempted. 2nd June: Cardiac dulness thought to be slightly diminished, *bruit* unaltered. On 24th July a systolic thrill felt

at the apex and reduplication of 2nd pulmonary sound. Towards end of July, although dulness was lessened, the *bruit* and accentuation were unchanged. Patient was sent for further rest to the Convalescent Inst.

Case 74. John M. W. 21, druggist, Ad. 30 May, 1899, Dis. 19 July. Reported by Registrar and Mr G. W. Gelderd.

Patient had shortly before been in hospital suffering from Raynaud's disease. His heart was then sound. On admission he was now complaining of pain in the left arm and back, which was thought to be rheumatic. Temperature normal. Cardiac dulness normal. A distinct systolic *bruit* is audible at the apex with accentuated 2nd sound at the pulmonary valve. Some headache. Unilateral sweats (on right side). 6th June: As the pains have not lessened the constant current was ordered. 20th June: Pain much relieved. Trophic changes are occurring in nails of left hand. All this time absolute rest, blistering between nipple and clavicle and administration of Sodium Iodide were being carried out. Towards the end of June the *bruit* became indistinct and on the 3rd July had finally disappeared. He was sent to his home in North Wales on 19th July being cautioned to avoid all exertion for some months. He was seen and examined on several occasions during the succeeding eight months and the heart was found to be perfectly healthy.

Case 75. John W. J. 33, tailor, Ad. 16 June, 1899, Dis. 19 Oct. Reported by Registrar and Mr P. J. Caddow.

Has had several prior attacks. Present illness began on 19th May. Pain and swelling in joints of hands and feet, profuse acid sweat. Temp. 101°. Cardiac dulness increased outwards and above. Soft systolic *bruit* at apex. 2nd sound accentuated at all areas, especially pulmonary. The mitral regurgitation thought to be of some duration. Uric acid in urine. A papular rash over front of thorax. Within a few days the pain in the hands abated, but it extended to elbows and shoulders. On the 29th all pain and also the rash had

disappeared; the systolic *bruit* at the apex remained without change. 3rd July: A relapse of the rheumatism. *Bruit* thought to be more distinct. 14th July: A second relapse of the rheumatism. Temp. 100°. 27th: All pain gone; a diastolic *bruit* heard down the sternum. 29th: *Bruit* at apex has assumed a "whiff" character. 31st: Distinct diastolic *bruit* at aortic cartilage. Pulse first noticed to be slightly collapsing in character. Pulsation in vessels of neck very marked. Capillary pulsation noted in lips. 23rd August: Cardiac dulness has increased of late and now extends to right of sternum and up to 2nd rib. Pericardial friction heard. Two more relapses of rheumatism have occurred. He gradually improved during September, and on Oct. 10th was able to get up. Quite free from pain. He has distinct aortic and mitral regurgitation. Discharged on Oct. 19th, the treatment having entirely failed to influence the condition of the heart.

Case 76. Rebecca H. 15, charwoman, Ad. 27 June, 1899, Dis. 25 July. Reported by Registrar and Mr Cook.

Had attacks of acute rheumatism three years ago and last January. A fortnight ago became markedly choreic. On admission, marked choreic movements, some conjunctivitis and a papular eruption (rheumatic?). No rheumatic pains nor any considerable rise of temperature. Cardiac dulness normal. Pulse 84. Systolic *bruit* at apex. Accentuation of 2nd pulmonary sound. No anæmia. The chorea was treated by arsenic and gradually disappeared; the *bruit* became soft and had entirely disappeared before 21st July. She went home on 25th, further rest being enjoined.

Case 77. Thomas B. 27, flagger, Ad. 11 July, 1899, Dis. 29 July. Reported by Registrar and Mr G. W. Gelderd.

Had severe acute rheumatism thirteen years ago. Present attack began three weeks ago. Pain in nearly all joints on admission. Temp. 102°. Cardiac dulness normal. No *bruits* on admission, but within two days a systolic *bruit* at the apex became manifest. The pain and pyrexia speedily disappeared,

and on the 24th the *bruit* had become inaudible. The patient, who is an obstinate and ignorant man, feeling himself free from pain insisted on leaving hospital on the 29th against advice. His heart sounds were quite normal.

Case 78. Margaret F. 16, factory girl, Ad. 12 Sept. 1899, Dis. 23 Oct. Reported by Registrar.

She was attacked by acute rheumatism in July of the present year. Prior to that date she had had no rheumatism and no dyspnœa. Pains in several joints. Temp. 100°. A systolic *bruit* at the apex on admission of unknown duration, with accentuation of 2nd pulmonary sound. Under treatment the pain disappeared and on the 29th Sept. the *bruit* had so far yielded to treatment that it was only heard when patient was recumbent. On Oct. 10th it was rarely heard even during recumbency, and on the 13th it was found to have vanished altogether. She was discharged on the 23rd Oct. with cautions to avoid exertion for some weeks, her heart apparently normal. Re-examined in January. Heart apparently normal. Examined again on 5th July, 1900. In good health. Working regularly in the factory. Has slight dyspnœa on ascending stairs or a steep hill. Cardiac dulness normal. No *bruit* at apex, but a suspicion of impurity about first sound. A certain amount of accentuation of 2nd pulmonary sound. When recumbent a systolic *bruit* is heard at the pulmonary valve which could be traced faintly at the apex. *Bruit* audible in the neck. Cephalic *bruit* also audible.

Felt some uncertainty as to whether the slight dyspnœa is due to hæmic causes, or to imperfect action of the mitral.

It is quite possible that the endocarditis had advanced too far on the 12th Sept. to admit of complete repair of the valve.

Case 79. Mary E. W. 18, no occupation, Ad. 24 Oct. 1899, Dis. 28 Dec. Reported by Registrar and Mr I. L. W. Nuttall.

Had acute rheumatism eight years ago. Sent in by Dr R. J. M. Buchannan, who had observed the onset of endocarditis a few days before admission. Several joints painful. Temp. 101·5°.

A systolic *bruit* heard with great distinctness at the apex. Accentuation of 2nd pulmonary sound. Pain soon yielded to treatment, *bruit* also softened and became inaudible on 6th Nov. 13th: Had a relapse. *Bruit* again heard with great loudness. 24th: *Bruit* is becoming soft: on 4th Dec. the *bruit* is only heard when patient is recumbent, or only rarely when she sits up. On 7th Dec. *bruit* ceased to be audible in any posture. On the 28th Dec. she was sent for further rest to the Convalescent Institution. Re-examined on 24th Jan. No *bruit*, no dyspnœa, but impulse was thought to be somewhat stronger than usual. Examined again on 28th Feb., heart perfectly normal.

Case 80. Matthew R. 25, joiner, Ad. 19 Dec. 1899, Dis. 26 Jan. 1900. Reported by Registrar and House Physician.

No prior attack. Pains in joints, fever and profuse sweating began at the end of November. Considerable pain on admission. Temp. 101°. Cardiac dulness normal. First sound at apex dull and prolonged. Soon manifested a systolic apex *bruit* and 2nd pulmonary became accentuated. On 18th Jan., the *bruit* became indistinct, and it disappeared a few days later, as also did the accentuation of the 2nd pulmonary sound. Went home on 26th being cautioned to avoid all active exertion for six or eight weeks. Was carefully examined on 5th June, 1900. Dulness normal. First sound clear and distinct at apex. No *bruit* anywhere. No accentuation of 2nd pulmonary sound. Patient is in perfect health, has no dyspnœa, and is working hard at his trade.

Case 81. James M. 21, carter, Ad. 9 July, Dis. 17 Aug. 1899. Reported by Registrar and G. W. Gelderd.

No evidence of previous attack. Ten days ago after a severe wetting had swelling and pains in many joints. Temp. 103°. Pulse 110. Respirations 40. Cardiac dulness increased in each direction, measuring six inches transversely. Pain referred to precordia, and tenderness in pressure over base of heart. Friction rub heard over sternum and left intercostal spaces.

A systolic *bruit* at the apex, conducted into axilla. Marked accentuation of 2nd pulmonary sound. The rheumatism and endocarditis were treated as usual, and over the blister a large poultice was used to allay the pericardial discomfort. 11th: Much better, pain in joints gone: breathing 32, easier: pulse 88, temperature normal. Precordial tenderness has gone, cardiac dulness an inch lower at upper border; friction still audible. 12th: Better again, pulse 80, respirations 24. Pericardial friction absent; sleeps well. 13th: Going on well, pulse 68. Respiration 16. 24th: Patient feels very well. *Bruit* still heard. Dulness diminishing. On Aug. 17th, patient who is an ignorant and self-willed person, not realising how serious his condition has been and is, refused to remain in hospital. The cardiac dulness is much diminished but the *bruit* is still unchanged.

Case 82. Mary A. G. 52, housewife, Ad. 12 May, 1899, Dis. 19 June. Reported by Registrar and Mr G. W. Gelderd.

No history of rheumatism until a month ago. On admission had pains and effusion in several joints, a vesicular eruption on the chest. Profuse sweats and a temperature of 102·6°. Cardiac dulness normal. No distinct *bruit,* but first sound at apex is indistinct and prolonged. Slight bronchitis and a slight pleuritic rub in the left side which disappeared early. Pains soon subsided under treatment, but first cardiac sound remained muffled and indistinct. On 23rd: a systolic *bruit* became apparent at the apex. This remained constantly audible for a fortnight; on the 8th June, the *bruit* was found to be vanishing and to disappear when she sat up. 12th: The *bruit* is now no longer audible in any position. 19th: Patient went home, being urged to rest as much as possible for some time to come. Her heart's action was normal.

Case 83. Mary C. 25, wardmaid in the Infirmary, Ad. 4 Dec. 1899, Dis. 8 Feb. 1900. Reported by Registrar and Mr Williams.

No definite history of previous rheumatism. Attacked at the end of November by rheumatic pains and severe peliosis rheumatica. Temp. 102·3°. On 8th December a systolic *bruit*

loud and distinct was discovered at the apex. Conducted to axilla. 2nd pulmonary accentuated. The peliosis gradually subsided. The *bruit* continued loud and strongly marked in all postures of the body until the 15th Jan. After that became softer and the pulmonary accentuation became less marked. Then followed a period during which the *bruit* was audible at some times and absent at others and always disappeared if she sat up in bed. On the 25th the *bruit* vanished altogether. 27th: The pulmonary accentuation no longer heard. On the 8th of February she went home with an apparently sound heart. Quiet and the avoidance of all muscular effort were strictly enjoined.

She was examined again in April and her heart found to be normal. She appeared well and had no dyspnœa.

Case 84. Jane G. 18, tailoress, Ad. 12 Dec. 1899, Dis. 10 Feb. 1900. Reported by Registrar and Clerks.

No rheumatism until last Sept. During December several joints were attacked. Temp. 101·2° on admission. She is rather anæmic. Cardiac dulness normal. Apex in normal position. Loud systolic *bruit* at apex, heard in axilla. 2nd pulmonary sound accentuated. A systolic *bruit* at the pulmonary cartilage, thought to be hæmic. 14th: Pain much better. 21st: Pain entirely gone, feels quite well. Apex *bruit* unchanged. 8th January: The apex *bruit* has now become so faint as scarcely to be heard. The pulmonary accentuation has disappeared, the aortic second sound now being the louder of the two. The *bruit* at the pulmonary cartilage is unchanged. If patient sits up in bed the apex *bruit* is not heard at all. 27th Jan.: Heart appears to be perfectly normal excepting as regards the hæmic *bruit* at the pulmonary cartilage. Went out on Feb. 10th, having been cautioned to avoid all exertion for some time to come. Her heart was carefully examined at intervals until the 24th of May. On that date she had every appearance of perfect health. Suffered no palpitation, had no trace of dyspnœa. Was able to carry weights and to ascend long flights of steps with ease. No hypertrophy, no *bruit* at any point. No accentuation of 2nd pulmonary sound.

Case 85. Lily P. 6, school girl, Ad. 2 March, 1900, Dis. 13 June. Reported by Registrar and Mr D. M. Alexander.

Has suffered from rheumatism. No pains at present, but is suffering from chorea. Temp. normal. Cardiac sounds normal, excepting a little accentuation of 2nd at the base. Dulness normal. 9th: Reduplication of 2nd sound at base: 23rd: Chorea is much less marked, but a distinct systolic *bruit* has become manifest at the apex; there is also marked accentuation of the 2nd pulmonary sound. 2nd April: *Bruit* seemed somewhat less distinct. 2nd May: Treatment has been continued diligently all this time, the apex *bruit* is now much softer and is becoming variable. 23rd: *Bruit* can be heard when patient is recumbent but not when she sits up. At the beginning of June the *bruit* finally disappeared, though some accentuation of the 2nd pulmonary still existed. Allowed to go home on 13th June, stringent orders being given that she be kept very quiet. On 10th July heart sounds normal, no dyspnœa.

Case 86. Charles L. 21, carter, Ad. 27 April, 1900, Still in Hospital. Reported by Registrar and House Physician.

No previous history of rheumatism. Attacked on 20th April by severe rheumatic pains. On admission Temp. 102. Many joints swollen and painful. Cardiac dulness normal, impulse seen in 5th space. A short, soft, but distinct systolic *bruit* heard at the apex. 5th May: Systolic *bruit* more distinct, no change in dulness. 14th: 2nd pulmonary sound accentuated. 21st: The *bruit* has been diminishing and is scarcely audible to-day, but the apex sound is prolonged. 26th: On sitting up no *bruit* is to be heard. 30th: Somewhat unwisely the patient was allowed to get up to-day. The *bruit* reappeared and was audible both when the patient is in the upright and in the recumbent position. Blisters and other treatment have been recommenced. June 7th: *Bruit* audible, slight accentuation of 2nd pulmonary sound. 21st: *Bruit* only heard when patient is recumbent; no accentuation now of pulmonary second sound. 25th July: Allowed to get up. No *bruit* anywhere, no dyspnœa. Quiet and avoidance of exertion enjoined for two months to come.

CHAPTER IX.

THE PRACTICE OF THE METHOD.

THE author is naturally anxious that others should employ these methods of preventing and of arresting valvular disease which he has advocated for many years. Some practitioners have already done so and have reported favourably of the results.

Two possible mistakes may be made in carrying out the method of treatment described, which must be carefully guarded against:

(1) It is important that suitable cases be selected; cases that are fairly recent. Those of course are the most satisfactory in which the date of the cardiac attack is known; but it more generally happens that the signs of valvulitis had set in before the patient is seen, and it is sometimes impossible to be sure in these instances what the duration of the attack has been. If the lesion chances to be five or six weeks old the chance is considerable that failure will be the issue. In such cases the practitioner must not blame the method of treatment, for it is only professed that the method will be of avail under certain conditions, of which a brief prior duration of the valvulitis is an essential.

(2) The second mistake is a lack of the required patience on the part either of invalid or physician. When the sick man feels no longer sick, when all pains

are gone, he naturally desires to get up, to return to his ordinary mode of life, and to his ordinary diet. Commonly in my experience of these cases some weeks pass without any improvement, and the lengthy confinement to bed is trying to both patient and doctor. The former usually does not think of the possibility of heart disease affecting himself, he was strong and active before and he fully expects to be so again; the physician does not like to cause alarm, but it not unfrequently becomes his duty to state in general terms and in as hopeful a spirit as possible what is the actual problem, viz. that the patient has been threatened with a form of heart weakness; that such often occurs during rheumatism; that in order to obviate risk it is desirable that he should remain absolutely at rest for a time; if he does that the probability being great that his recovery will be perfect.

On the whole I have more fear of the doctor's patience when week after week passes and the *bruit* remains without change. He, especially if he is new to the mode of treatment, is very apt to lose heart, to say to himself "We have tried this plan for two or three weeks and not a trace of improvement has shown itself, it is not worth while keeping this poor fellow in bed any longer." The author himself has often felt hopeless and been inclined to abandon further care in cases which in the end have proved the most perfect of successes.

When this temptation has been resisted the physician and the former invalid have often had occasion subsequently to remark how well worth while it was for the latter to remain for a few extra

weeks in bed and under treatment, in order to secure a perfectly sound heart and the ability for exertion and enjoyment.

The boon of a sound and normal heart is indeed so great that it is worth almost any trouble and any sacrifice to retain it.

I confess that few things give me more pleasure than to observe day by day the gradual waning, and final disappearance, of all signs of valvular mischief in a patient in whom its commencement has been only too distinctly manifest. To send him home with a sound heart, and from time to time to test and observe the circulatory mechanism, to make sure that all has gone on favourably, is one of those gratifications which compensate the physician for much that is painful and trying in his professional experience.

To me it certainly is a pleasurable and a comforting thought that out of many hundreds of cases of acute rheumatism treated in my wards during the past fifteen years, only three, to the best of my knowledge, have left them with heart disease actually acquired while under my care.

It is also a great satisfaction to observe that several distinguished writers on Diseases of the Heart are now pointing out the importance of prolonged rest and other preventive measures in commencing endocardial disease. Among them are Sir R. Douglas Powell, who in his excellent treatise on the *Principles of Treatment of Diseases and Disorders of the Heart*, published in 1899, strongly recommends prolonged rest, and the administration of Iodides[1]. Dr G. A. Gibson in his *Diseases of the Heart and Aorta*, 1898, speaks

[1] pp. 52—53.

favourably of the method advocated in these pages[1]. Dr Cheadle, in his article on "The Acute Rheumatism of Childhood" in Allbutt's *System*[2], speaks strongly of the importance of absolute rest and of the need to avoid excitement and chill. Prof. Whittaker in his article on "Heart Disease" in the *Twentieth Century Practice of Medicine*, speaks of rest as the most essential element in treating this condition and also of the importance of a light diet[3]. Dr Byrom Bramwell in his treatise on *Disease of the Heart* speaks of the necessity of a rigid system of rest in the treatment of early acute endocarditis[4].

In the author's opinion the time is much to be desired and hoped for when every practitioner shall regard each case of acute or subacute rheumatism not as rheumatism only, but first and before every thing else as a case of *impending peril to the heart*, endangering the whole future life of the patient. The mere rheumatism *per se*, the pain, fever, and general discomfort, with the means at our disposal, we can usually soon mitigate, and the patient will, after a few weeks' care recover; all this is quite secondary to the great question, will the heart escape damage? Our energies ought to be mainly directed to the endeavour to give the heart the best chance we can to maintain or to regain its normal function, with a strong hope of success if our efforts are begun sufficiently early. There is no lack in our profession of the desire to prevent valvular disease, there is only the lack of belief in its possibility.

[1] p. 426.
[2] Vol. III. p. 52.
[3] Vol. IV. p. 183.
[4] p. 393.

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